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BRITISH MEDICAL JOURNAL

LONDON SATURDAY NOVEMBER 12 1949

TUBERCULOSIS IN EUROPE DURING AND AFTER THE SECOND WORLD WAR

BY

MARC DANIELS, M.R.C.P., D.P.H.

Member of the Scientific Staff of the Medical Research Council

Among the suggestions made by Dr. Milroy for the lectures on State medicine and public health which he established over 60 years ago is the following :

"XVIII . . . that more diligent and continued attention may be paid than has yet been attempted in this country to the study of the accurate *Geography of Diseases*, (and to) the exact date of each epidemic prevalence . . . of the ordinary endemic maladies in different countries and localities, together with a brief notice of any exceptional feature . . . characteristic of each aggravated manifestation of the malady in question, so that some record of its varying natural history may be preserved for the benefit of future times. The want of thoroughly trustworthy information on this department of professional investigation has had much to do with causing the numerous errors and fluctuations of opinion which have hitherto retarded the real progress of sound knowledge on various questions. An accurate geography of many diseases is often essential to the complete determination of their true natural history."

It is a sobering thought that these words, written in days when the epidemiological study of infectious diseases was still in its infancy, are as true of tuberculosis to-day as they were then. The amount of work that has been done on tuberculosis, and particularly the amount that has been published in the past 50 years, defies calculation ; but the number of sound epidemiological studies of the disease can easily be enumerated, and our knowledge of its natural history is still rudimentary. In fact, though there has been a progressive and striking reduction in tuberculosis mortality since the time of Dr. Milroy, it is still the main killing disease at the most productive ages of man. Dr. Stocks (1944) has estimated on the basis of a combination of morbidity and mortality statistics that in 1938 there must have been at any one time in England and Wales about 100,000 persons with clinically active pulmonary tuberculosis and about 650,000 in a quiescent state ; and of this total about 60,000 were expected to die eventually of the disease. Its effect on the productive capacity of the population is demonstrated by the fact that the mortality from the disease in England and Wales in 1945 represented a loss of 565,000 person-years of future lifetime between the ages of 15 and 65 (Stocks, 1949).

The nineteen-thirties and the early years of the war saw discoveries of so great importance in the treatment of acute infections and in the prevention of such diseases as typhus that they reduced to relatively low proportions the toll usually taken by war in disease. It had become a commonplace to compare the numbers who died in past wars from casualties in battle with those dying from diseases such as typhus, dysentery, and typhoid. But for the second world war this does not apply ; though there were some epidemics of these diseases they did not reach anything like their former gravity and distribution. Tuberculosis rates, however, rose in every European country seriously affected by the war, and there is now a growing realization in Europe that tuberculosis is our main public health problem. A study of the trends of the disease during the war years may be of some value in contemplating future action.

With these considerations information is presented here on wartime and post-war tuberculosis trends, and on the development of tuberculosis services in different European countries since the end of the war. Much of the information was collected during service with Unrra ; the disturbing scenes one saw then aroused and focused interest on some of the problems to be discussed here.

DEATH RATES AND PREVALENCE*

Tuberculosis Mortality

When one presents statistics of tuberculosis in Europe it is with a feeling that they are better than no information at all, but not much better. There are countries in which as many as 50% of the deaths are not medically certified, and where 15 to 20% are registered as being due to unknown causes. Even in France as many as 10% of deaths are registered as of cause unknown. Most regional statistics within the European countries are unreliable ; in most cases the deaths are registered at the place where they occur and are not referred to the residential population. The fear of tuberculosis in backward populations is responsible for much evasion of tuberculosis certification. In addition to all this, one must take into account the many factors operating during war and the German occupation, conditions to make statistics of any kind many times less reliable than before. The drafting of men into military service or into forced labour for the Nazis, the Jews deported, the numbers taken prisoner of war or thrown into concentration camps ; the people who died away from their homeland by violence on the battlefield, in air raids, or in gas chambers, or of untreated diseases in camp or elsewhere ; the migration of millions during and after the liberation, including the return to their homes of prisoners of war many of whom had a high incidence of pulmonary tuberculosis : these factors combine to modify all statistics

*The first of two Milroy Lectures (abridged) delivered at the Royal College of Physicians on February 15 and 17, 1949.

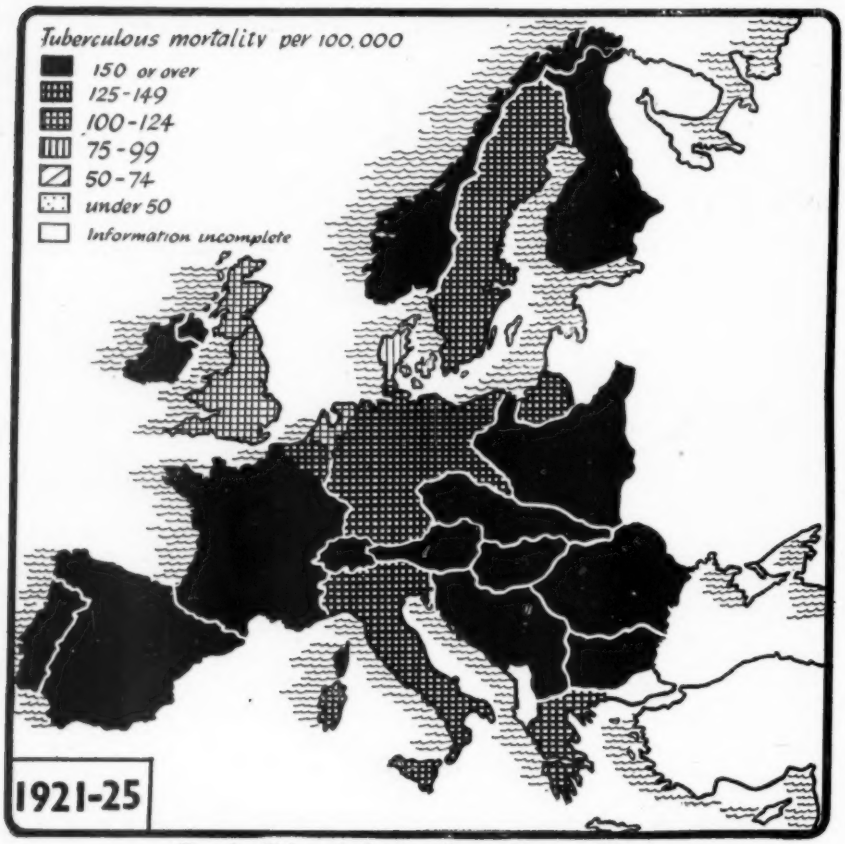


FIG. 1.—Tuberculosis mortality in Europe, 1921–5.

obtained. So all the figures to be given here bear this reservation : they are the statistics that were obtainable in the circumstances, the circumstances of war and post-war.

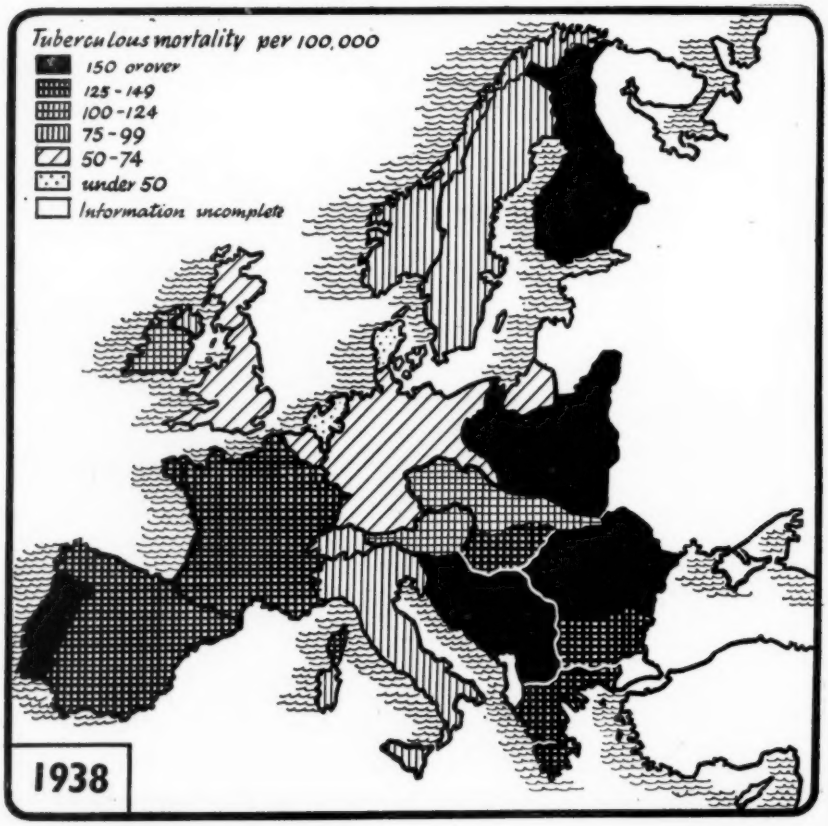
Before looking at the trends of tuberculosis mortality during and after the second world war we can rapidly view the remarkable fall in the rates over the past 50 years. The tuberculosis death rates fell in several European countries from above 200 per 100,000 in 1885 to below 100 in 1935. The most striking fall was in Denmark ; the rate there was one of the highest recorded in Europe in 1885, with a mortality of nearly 300, and in 1935 it was below 50. In most European countries, and particularly in Germany, Austria, and Belgium, there was a sharp rise between 1914 and 1918. A striking feature of the trends is that shortly after the end of that war the rate had declined to a level which it would normally have reached if the pre-war trends had continued uninterrupted.

The changes in the period between the two wars and during the recent war are demonstrated in the maps (Figs. 1–3). In Fig. 1 are charted the tuberculosis mortality rates in the countries of Europe in the period 1921 to 1925. It is obvious from this map that tuberculosis death rates in European countries were in the nineteen-twenties still very high according to present standards in all countries except Denmark ; the mortality was highest in Eastern Europe, France, Spain, Norway, and Ireland.

The second map (Fig. 2) demonstrates the striking change which had taken place by 1938. In England and Wales, Scotland, Belgium, Holland, Denmark, and Germany the tuberculosis mortality had fallen below 75 per 100,000, and in Norway, Sweden, Switzerland, and Italy it was below 100. In France, Spain, Portugal, and practically the whole of Eastern Europe the rates were still very high, though in Czechoslovakia and in Austria there had been some improvement. It is this particular picture that we must bear in mind in considering to what extent the tuberculosis situation was affected in the different countries by the conditions of war.

In Fig. 3 are charted the tuberculosis death rates for 1946. Statistics for some countries are as yet so incomplete that it has been necessary to leave them blank, but an outstanding feature is the relatively low rates in the whole of the mid-continent of Europe : in France and in Norway and Sweden the mortality was lower than in 1938, in Germany and Scotland somewhat higher. We have figures as shown for Poland, Rumania, Hungary, and Bulgaria, and it is justifiable to assume that the tuberculosis mortality in the other Eastern European countries is as high as in these. Before the war the rates in these countries were among the highest in Europe.

FIG. 2.—Tuberculosis mortality in Europe, 1938.



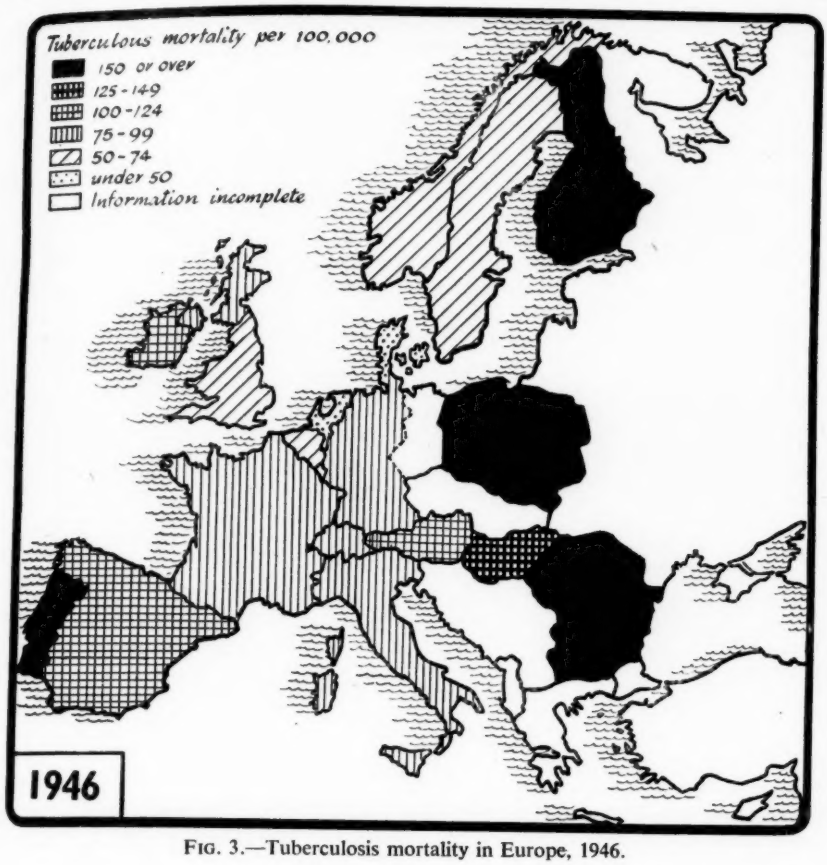
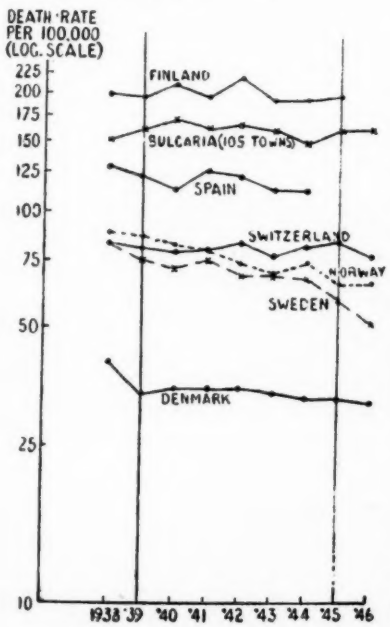


FIG. 3.—Tuberculosis mortality in Europe, 1946.

Coming now to a study of what happened in each country during and after the war, we can consider three main groups of countries : one in which there was little or no wartime rise in tuberculosis mortality, a second group in which there was a rise in the early years of the war and then a continuous fall, and a third group in which there was a rise in the latter part of the war.

FIG. 4.—Tuberculosis mortality in European countries having little or no wartime rise, 1938–46.



I. Countries in Which there was Little or No Wartime Rise (Fig. 4)

In Denmark the rate was low in 1939 (rate 34) and slightly lower in 1945 (rate 33). It has the lowest tuberculosis mortality in Europe. At the beginning of the century the rate was as high as in most other countries. In the past 50 years there has been a very notable rise in living standards in this small country, and during the period between the two wars public social care was developed considerably. In this war Denmark fared better than most countries ; certain food supplies were limited, but there was never an acute shortage. In a number of other countries with particularly high tuberculosis rates before the war the rates remained high but there was no significant change, and Spain, Bulgaria, and Finland are typical of these. Switzerland had a medium rate, and, though there was an arrest in the decline of the pre-war years, there was no serious rise. Finally, in two countries, Norway and Sweden, there was a definite fall during the war years—in Sweden from 82 in 1938 to 51 in 1946, and in Norway from 88 to 65. There was a rise in 1943 and early 1944 in Oslo, attributed to a deterioration in the food supply.

II. Countries in Which the Rate Rose in the First Years and Fell in the Later Years of the War (Fig. 5)

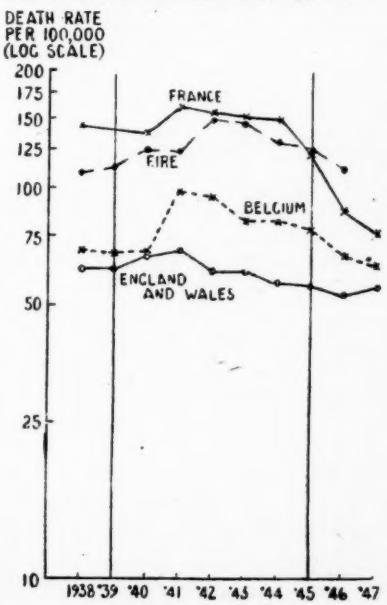
The principal countries showing this trend are England and Wales, Belgium, Eire, and France.

In Belgium the rate rose from 69 to 98 in 1941 and fell subsequently to a level in 1946 just below that of 1938, and there was a further slight decline in 1947. There is enough in the chaos of the first two years of the war to explain the rise to a peak in 1941 : the destruction and occupation of sanatoria, the discharge of patients to their homes, the breakdown of food distribution, the overcrowding and lack of hygiene. However, the central tuberculosis organization was re-established rapidly, after some opposition from the occupying authority, and by 1942 there were almost as many sanatorium beds available as before the war. Double rations were accorded by the dispensaries not only to patients with acute tuberculosis but also to those whose lesions had been active within the past five years. All things considered, there is enough to explain the rapid recovery after 1941, though it is important to note that the pre-war level had only just been reached in 1947.

In Eire there was a continuing rise until 1942–3 ; apparently Eire was the only neutral country in Europe to suffer from wartime conditions to this extent.

The death rates for France show an interesting phenomenon. The curve for the whole of France shows a rise in 1941 and a subsequent decline, at first gradual and then quite steep. The rate now is little more than half that for 1938. These trends were not the same throughout France. The curve for Paris shows the

FIG. 5.—Tuberculosis mortality in European countries in which the wartime rate rose and later fell, 1938–47.



general trend in an exaggerated form, with an acute rise from 155 in 1938 to 215 in 1941 and subsequently a very rapid fall to a level far below that of 1938 (72 in 1947). But other regions showed quite different tendencies. In Normandy, Brittany, and Eastern France there was an ample supply of animal food and milk, and in these regions, where there had always been a high mortality from tuberculosis, the rate declined throughout the war. In the South, on the contrary, in Bouches du Rhône, in Vaucluse, and in Var, the tuberculosis problem continued to be acute up to 1943, and this can be related to the very severe food shortages in these districts.

For France one must make reservations on the absolute value of mortality figures, but comparisons between years are valid. Apart from the striking feature of the differing reactions in various parts of France, the most outstanding is the very rapid decline of the death rate in France after the preliminary rise. This fall in the tuberculosis death rate is greater than for any other country in Europe. During the first years of the war the tuberculosis and the general death rates followed a similar course. But since 1943 the general death rate has remained higher than before the war; so the proportionate tuberculosis mortality in the department of Seine, for instance, went down from 13.5% in 1938 to 9.1% in 1945. For the whole of France it is now 6%, and this means in fact that it is approaching the level of relative importance that tuberculosis has in England as a public health problem. If we consider the pulmonary tuberculosis mortality separately we find an even more acute decline; the death rate from non-pulmonary tuberculosis was unchanged. It is important to note that this steep decline in the death rates from tuberculosis has been observed in most parts of the country; this is obvious in the most recent figures for separate large cities of France.

In England and Wales, as in Belgium and France, there was a rise in the first two years of the war, though less acute than in those countries. Obviously our experiences were less severe from many standpoints than those of the occupied countries, but in 1940 the air raids and the anticipated invasion, with evacuation of sanatoria from the danger areas and of hospitals to make room for air-raid casualties, combined to produce a situation highly favourable for the tubercle bacillus. The increase was 13% in the first two years. In this increase anxiety and overwork must have played an important part; there is evidence also that increased opportunities for infection were a major factor. The evacuation of hospitals and sanatoria, the black-out conditions, the overcrowding in homes because of air raids and billeting, the numbers of tuberculous patients drawn into industry, and the coming in contact with them of numbers of people who previously had led a relatively isolated life, must all have combined to increase infections greatly over the pre-war rates. There was in fact a marked increase among children under 5: between 1938 and 1941 deaths from respiratory tuberculosis went up 55% in boys and 105% in girls.

For meningitis in children up to 10 years there was an increase of 50%, and there is evidence that the majority of these infections were caused by the human bacillus. In mental hospitals in England and Wales, which already before the war showed an alarmingly high tuberculosis mortality—namely, 377—the rate increased by 140% to 901. Here the dominant factors must have been low nutrition and increased infection with overcrowding. Since 1941 the tuberculosis death rates in England and Wales have gradually declined, though they have not reached the level that would have been expected if pre-war trends had continued uninterrupted. In children under 5 the death rate has remained above the level reached before the war.

III. Countries in Which the Death Rates Rose Through the War Years to a Peak at the End of the War

In Fig. 6 are shown the trends in several countries; the rates are those for large cities, as with the exception of Holland the overall national rates are not known. All show

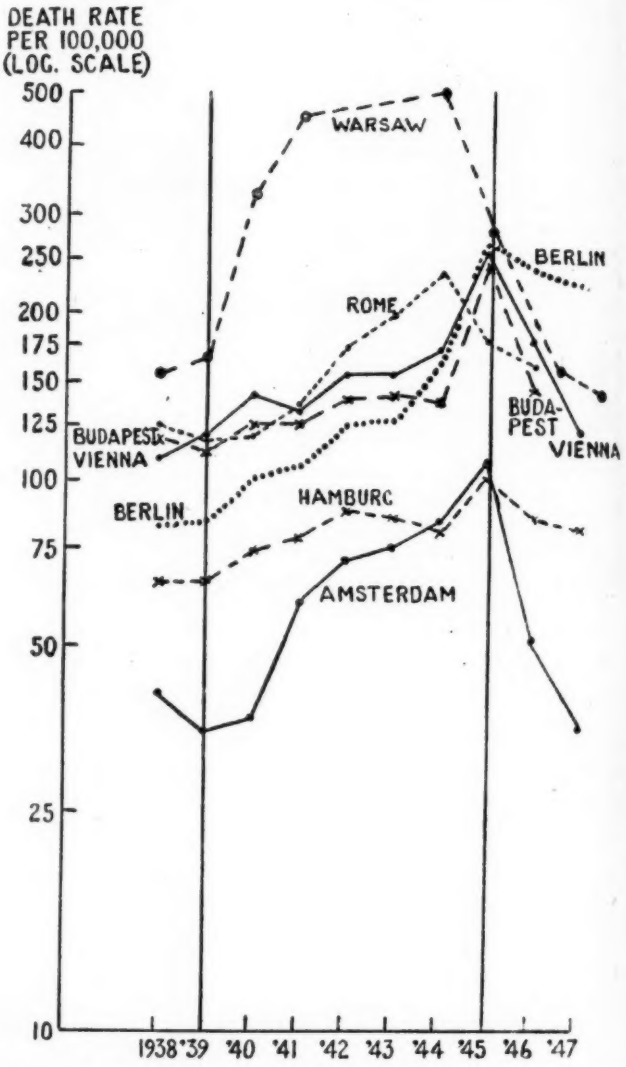


FIG. 6.—Tuberculosis mortality in European cities, 1938–47.

the same type of curve with an acute drop at the end. All are countries which were most severely affected in the final years of the war.

In Holland the rise was greater in Amsterdam than in the rest of the country—this is the more important as between the wars the rate in Amsterdam had declined to a lower figure. Between 1939 and 1945 it rose 208%, compared with the rise of 111% for the whole country. There is no doubt that the worst suffering in Holland was during the fighting in the final year. In Amsterdam many died of starvation. The deaths from all causes in the first half-year, which in 1939 numbered 3,736, totalled 9,701 in 1945. Deaths from infantile diarrhoea were 4 in 1939, 38 in 1944, and 255 in 1945.

In both Hungary and Austria there was a very acute rise at the end of the war. In Budapest the food situation

was not bad during the war, but there was chaos during the final year, and inflation with an acute food shortage. A very similar situation developed in Vienna. The highest mortality in Vienna was in the working-class boroughs ; in one of the boroughs where there were the worst housing conditions the death rate was 322 in 1945. In both cities there was a sharp drop after 1945.

For Rome the peak of mortality was in 1944, and the death rate subsequently fell steeply. A study of the mortality in different parts of Italy reveals some interesting factors, reflecting here as in France the differences in conditions. The trends up to 1942 are very similar ; by then there was an increase in tuberculosis deaths of 36–41% for respiratory tuberculosis, 21% for other tuberculosis. For cities of Southern Italy the mortality then went down gradually ; we can recall that this part of the country was little affected by the fighting after the invasion. In Northern Italy there was a fall in 1943 and 1944 and another rise in 1945 corresponding with the campaign at the end of the war. But in Central Italy, where the most severe war conditions prevailed and for the longest period of time, the death rate rose steeply in 1943 and 1944 to a rate 130% above that of 1939. Subsequently there has been a drop, but in 1946 the rate (83 for the whole of Italy) was still above the pre-war figure : if the rate of decline in pre-war years had continued, mortality in 1946 would have been about 50.

In Germany also conditions were worst in the final years of the war ; the aerial bombing, the disruption of public health activities, and the breakdown in food supplies must have contributed to produce the acute rise in 1945 after a moderate rise in the preceding war years. In all areas there has been a fall in mortality since 1945. The situation is much graver in Berlin than elsewhere. Among reasons given for the greater mortality in Berlin are : during the war, the effects of continual as opposed to sporadic raids, and since the war the food difficulties—much more serious than in the British and United States Zones—and particularly the fact that Berlin has been cut off from use of its sanatorium beds, most of which were outside the city. In this respect the Zones were relatively well p'aced ; Berlin was left with a little over 5,500 beds, or 0.7 bed per death, compared with over 2 beds per death in the British Zone. The exceptional opportunities for hospital treatment of cases in the Zones may well be responsible for at least a part of the post-war decline in mortality. The tuberculosis mortality in the Western Zones of Germany is well below that of many other European countries, including Scotland and Ireland. The most recent figures for 1948 show a continuing fall in all areas, including Berlin, where the rate for the first half-year was 30% below that for the first half of 1947.

Poland is one of the countries which already before the war had a high death rate from tuberculosis. Figures for the whole country are not available, but we know that in the cities with one exception the rate was over 150 per 100,000. Despite wholesale destruction by the Germans, some records were preserved, and it has been possible to trace the course of the disease through the grim years of the occupation. Already by 1941 the mortality from tuberculosis in Warsaw had increased by nearly 200% over 1938, and in Lodz the increase was 80%. These are the worst recorded tuberculosis death rates that we found anywhere ; there is sufficient in the conditions of the occupation to explain the magnitude of the rise. The rate persisted at a very high level, increasing further until the end of the war, since when there has been a rapid fall, and in all towns the rate is now well below the pre-war level. For combined figures of six main towns the most recent rate—for the year from mid-1947 to mid-1948—is 28% below the 1938 figure. In Katowice, a flourishing mining and industrial centre, the rate is now 63 compared with 101 pre-war.

There is less information on the mortality trends in Yugoslavia, but in the city of Ljubljana, which before the war had a particularly active tuberculosis control department, the death rate rose from 70 in 1940 to 263 in 1944.

Among the millions who died in concentration camps tuberculosis was only one of many causes contributing to death ; but it was found in a very high proportion when the conditions in the camps were revealed at the liberation : 40% of necropsies at Dachau showed advanced tuberculosis. Of 64 cases of starvation studied in Belsen Hospital 27 had definite tuberculosis. At Buchenwald, Dachau, Nordhausen, and other camps, hundreds of tuberculous patients were taken out when the camps were liberated, many of them so ill that they died within a few days.

Tuberculosis trends in Scotland merit particular study, as this is the one country which, after having been seriously affected by the war, has shown no improvement in rates since the end of the war : there has even been a rise to a rate higher than the peak during wartime. Fig. 7 shows the death rates from respiratory tuberculosis between 1932 and 1946. The curve for the whole country shows the slow decline between 1932 and 1939 and the rise in the first year of the war; this level was maintained to 1945, since when there has been a further rise to a rate higher than in 1932. The combined rate for the large burghs shows a marked rise, and maintenance at a level well above 1932. The rate for the counties (excluding large burghs) showed a steeper decline in the pre-war years, a less marked but sustained increase during the war years, and in 1946 a rate still below the 1932 rate. The main part of the rise in Scotland is due to the rates in the cities, and it is interesting to look at a few of these specifically. Fig. 8 shows the death rates from respiratory tuberculosis from 1920 to 1947. In Aberdeen there was a fairly consistent fall through out the nineteen-twenties and nineteen-thirties, a rise during the

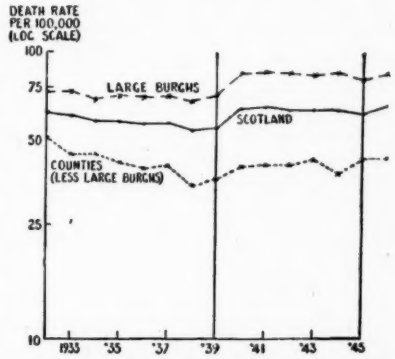


FIG. 7.—Death rates from respiratory tuberculosis, Scotland, 1932–46.

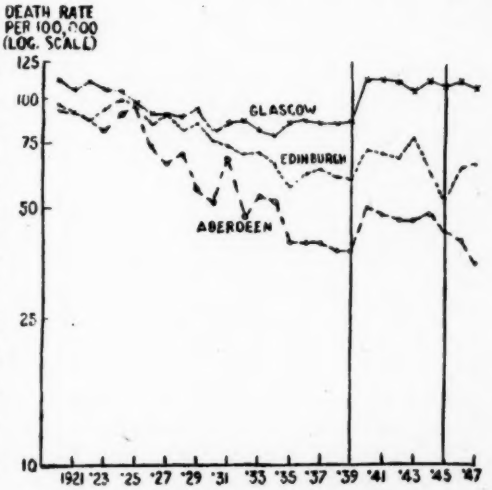


FIG. 8.—Death rates from respiratory tuberculosis, Scottish towns, 1920–47.

war, and a fall since then to a level below that for 1939; this is similar to what is happening in England. In Edinburgh there was a slower decline in the pre-war years, a fall at the end of the war, then a rise. It is Glasgow which presents the main problem: with its population of one million—i.e., one-fifth that of the whole of Scotland—it plays a large part in determining the overall trends. The first striking fact is that the decline in tuberculosis rates was arrested during the nineteen-thirties. Secondly, the wartime rise was steep and has been maintained at a level as high as the 1921 rate. There is at present a commission studying the causes of these trends, and one can only underline the differences between the trends in Scotland and those in the rest of the continent of Europe.

Age and Sex Analysis of Death Rates

An interesting phenomenon in the wartime rise in tuberculosis has been the predominance of the rise in men, except in Scotland (Fig. 9). In some countries, in fact, the rise was confined to males. Even in Denmark, where the overall rate from pulmonary tuberculosis had remained stationary at the low rate of 29 throughout the war, analysis shows that this rate was composed of a rate in males rising from 28 to 33, and in females falling from 30 to 25. Similar trends were observed in Holland.

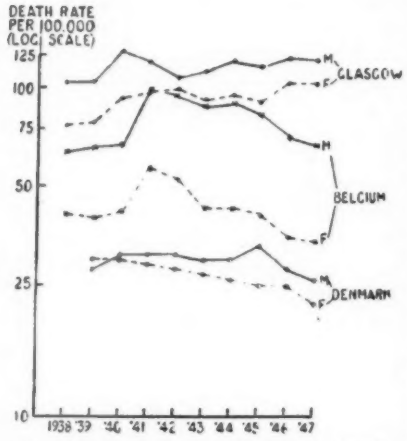


FIG. 9.—Respiratory tuberculosis death rates (male and female); showing predominance of the rise in men in Belgium and Denmark, compared with the trend in Glasgow.

In Belgium, where as in most other countries the rate in males was already higher before the war, the phenomenon is more striking. In 1941, at the peak of mortality, the increase over the 1938 rate was 49% in males and 36% in females. The subsequent decline was much slower in males: in 1944 the female rate had returned to the pre-war level, and by 1947 was 20% below; the male rate in 1944 was still 32% above the pre-war level, and did not reach it till 1947. There is much less difference between the sexes in the rates for non-pulmonary tuberculosis. In Italy there was a similar rise; whereas in 1939 the male death rate was 2% higher than in females, in 1946 it was 27% higher.

In France there was very little rise in the female rate in the years 1939–41, and a considerable drop subsequently at all ages. In males there was a very great increase at all ages, and a subsequent drop much below the 1935 rates. It is particularly marked in the 30–50 age group. The overall rise in the first two years of the war was 16% in females and 57% in males. A noteworthy feature of the remarkable fall in tuberculosis mortality in France is that it occurred in all age groups except young children and old people.

In Berlin, where as we have seen there was an alarming increase in tuberculosis, the increase was greater in men than in women, and occurred at all adult age groups. In Hamburg, if one compares the 1946 rate with that for 1939, in females there was no increase, whereas in males the rate was nearly 50% higher in 1946 than in 1939.

In Scotland, on the contrary, there has been a greater increase in females than in males (in 1946–7 the respiratory tuberculosis death rate was 19% higher in males, 32% higher in females, than at the outbreak of war). In fact, the exceptional rise of rate in women is the main feature of the persisting high rate in Scotland when compared with the rest of Europe. An analysis of the data in age groups shows that in females the increase was predominantly in young women. In men the increase was wholly in the older age groups.

Many possible causes have been suggested for the excessive rise of mortality in males in countries other than Scotland. Certainly in part it may be due to the selection by the Services of healthy non-tuberculous members of the young adult population. But where rates are calculated on the whole population this does not apply; and, moreover, we have seen that the older age groups showed a greater rise than those affected by military service. Deterioration in working conditions and participation in outside work in civil defence duties in this country or in resistance movements in other countries may have played a part. In this respect, however, it is interesting to note that in the mental hospitals in England and Wales the increase was greater in males than in females and that it has persisted in males at a level 60% above 1939, whereas in females it is now back at the 1939 level. There, either added exposure to infection or undernutrition, or both, must be responsible. It has been suggested elsewhere that reaction to malnutrition is more severe in males than in females, but definite proof of this is lacking.

It is interesting to look in rather more detail at the age-group trends in each of the sexes. In Fig. 10 for England and Wales, where the rates are adjusted to include non-civilians, one sees first that in females a peak at ages 20–24 is maintained, and that there is a fall in all age groups, whereas in males the rates are lower in 1947 than in 1939 at all ages except young children and the over-fifties. This is part of a phenomenon which is being increasingly noted in other countries—namely, the maintenance of the acutely peaked curve in females with a maximum at ages 15–24, though with falling rates, and in males the rounded curve with high rates at all adult ages but a peak gradually shifting towards the older age groups. This phenomenon of the

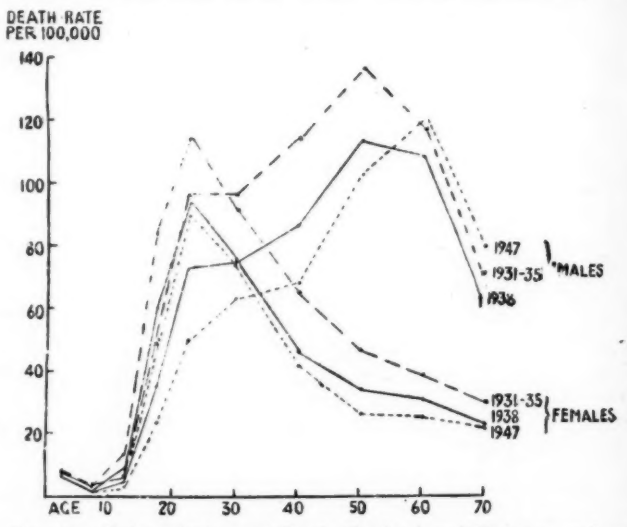


FIG. 10.—Tuberculosis mortality in England and Wales at various ages (male and female, and including non-civilians), in 1931–5, 1938, and 1947. Respiratory tuberculosis only.

shift of the peak to the older age groups in males has given rise to various hypotheses. A most interesting one is based on a statistical analysis of tuberculosis mortality in successive generations: the mortality in different age groups plotted on a generation basis shows little change in the type of curve through successive decades (Springett, personal communication).

Tuberculosis Morbidity

I have taken the tuberculosis death rate as the main index of tuberculosis trends. At present it is the most reliable index. Statistics regarding new cases diagnosed in dispensaries each year, or new notifications, or total cases known to the dispensaries at any one time are not lacking. But in the conditions of war these are practically valueless for comparative purposes, either for comparison between successive years in the same country or between different countries. Fig. 11 is characteristic. It shows for Belgium the dispensary case rate going up to a very high figure at a time when the death rate was well on the way down. Had that rise represented a true rise in morbidity there would have been a delay in the decline of the death rate or at least another rise later on. But it is clear from the accounts by the Belgian authorities themselves that, with the granting of double rations for the tuberculous at a time when rationing was very severe, the dispensaries were beset with people more or less asking to be diagnosed as tuberculous—and in the conditions of the occupation there was little reason why doctors should refuse. In France, too, the notification rate continued to rise at a time when the death rate was falling: in 1945 the total of new cases on the register was 45% above the 1938 figure, but the death rate was 20% below, and subsequently it continued to fall steeply.

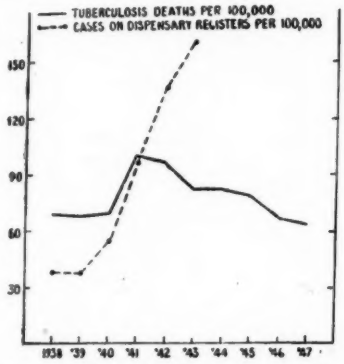


FIG. 11.—Morbidity and mortality statistics, Belgium, 1938–47.

The problem of assessment of tuberculosis prevalence by the morbidity rates has arisen again in the past two years in Germany. Alarming reports were issued pointing to a very acute rise in the numbers of notifications. It was said that, though the death rate was declining slightly, another rise would certainly occur. However, the death rate has continued to fall.

Thus even in the few countries in Europe where notification of tuberculosis is compulsory there may be very serious causes of error. The morbidity statistics may be of value in a country where tuberculosis is notifiable, where there is a well-defined and constant basis of notification, where the economic situation is not such as to influence the diagnosis, and at a time when new methods of examination are not being introduced. It is possible that much information may be derived from them in the future.

Results of mass radiography could conceivably provide more valid information concerning the prevalence of tuberculosis. No comparison can be made with pre-war prevalence, but comparison should be possible between different European countries, since, when properly employed, results should indicate what proportion of a community at any single time have lesions of pulmonary tuberculosis significant enough to require observation or treatment. The method is being extensively used now for the early detection of tuberculosis, and it is tempting, despite variations in diagnostic interpretation, to compare in detail the results obtained in different countries. There are some gross differences that one cannot ignore—between on the one hand the Scandinavian countries, France, Germany, and this country, with an incidence of about 3 to 5 per 1,000 of active tuberculosis requiring treatment, and, on the other hand, countries like Greece and Poland, with an incidence of corresponding lesions in 10 to 50 per 1,000 of the populations examined. Mass-radiography examination of Frenchmen returning from Germany in 1945 revealed pulmonary lesions in 1.5% of prisoners of war, 2.5% of men coming from labour camps, and 4.4% of men from concentration camps. There can be no doubt from present-day surveys that there is a very high prevalence of tuberculosis in Eastern European countries. At the present stage of development of this method, however, it is probably not justifiable to make more detailed comparisons.

Statistical Analysis. Conclusions

Looking back over this collection of statistics, there are at least three outstanding features about which there can be little doubt, despite the reservations regarding the absolute validity of some figures. One is that the wartime rise was closely related to the severity of war conditions; the second that the rise predominated in men; and the third that in nearly all European countries there has been an important fall in tuberculosis mortality since the end of the war.

The study of trends in different countries has shown that the increase in tuberculosis was related to deterioration in conditions so closely, in time and in gravity, as to constitute a remarkable index of the wartime tribulations of those countries. That tuberculosis prevalence is closely related to living conditions and nutrition is no new conception and has been shown in several studies of under-privileged communities. The incidence of tuberculosis discovered by mass radiography in Polish university students is on the same level as the incidence of lesions discovered in homeless destitutes of New York City (Edwards, 1940).

In some ways the disaster was not as great as in previous wars; this applies particularly to the countries where public health services had developed to a high degree between the two wars. In former years the health and social services in peacetime were barely adequate to maintain a perilous state of equilibrium, and the disaster of war would so upset the balance as to produce chaos. This time, in countries such as our own, after the first crisis with the evacuation of hospitals the health and social services were rapidly re-established, and measures designed to protect the people's health were extended well beyond those of peacetime. It is to the credit of those responsible for planning the health and food services of the country during and after the war that the wartime attack on health was relatively well resisted. On the other hand, where the social and health services were relatively undeveloped before the war, where already before the war there were very high tuberculosis rates—and this applies to many of the Eastern European countries—the wartime reaction was nothing less than catastrophic, and was made even more so by the conditions of the German occupation.

There has been much conjecture regarding the nature of the wartime rise. The additional deaths could be of patients who under normal conditions would have lived a little longer; they could be of those patients who would normally have overcome their disease and survived. In both events the main factor responsible would be the

deterioration of living conditions and of nutrition aggravating the prognosis of established disease. Or, thirdly, the deaths could be of persons who were being infected for the first time in greater numbers than normally because of increased exposure. No doubt in most countries there was a combination of all three factors.

In this respect the post-war fall, and in particular the rate of fall, is worth studying. Were the wartime rise due solely to the premature death of patients suffering from the disease we would expect a steep rise followed by a deeper fall, and it is possible that this may occur where conditions are severe enough to precipitate the death of patients with a bad prognosis, but not to provoke any great extension of the disease. This may partly explain the remarkable fall in France, but other factors must intervene also.* In nearly all other countries, where the pre-war decline was resumed at the same level or a higher level than would have been observed normally, it is obvious from the plotted rates for both world wars that there were many thousands of deaths from tuberculosis which would not have occurred but for the war. To what extent these were due in the main to aggravation of disease from which the patient normally would have recovered, and to what extent to new infections, must remain an open question.

We are far from a complete understanding of the nature of the wartime and post-war trends in tuberculosis. The uncertainty of our conclusions regarding widely differing trends, in for instance France, England, and Scotland, underlines the need for sounder and more detailed epidemiological study of tuberculosis in the future. Such study is at any time indispensable to a rational programme for eradication of the disease; it is particularly so at a time when our resources are limited, and action must be directed towards those groups of the community revealed in this way as being most susceptible to the disease. It will certainly be indispensable in the future when improvement in social services reaches the limit of reduction of tuberculosis by such means and we are left with the need for a real understanding of the disease if any further progress is to be made.

[The second lecture, with a list of references, will appear in our next issue.]

*One possible factor is the fall in consumption of alcohol since the introduction of rationing of wines and spirits in France during the war. This was followed by a marked fall in morbidity and mortality from diseases attributable to alcoholism, a fall not paralleled in any disease other than tuberculosis.

A residential college—the Midwife Teachers Training College—has been opened for midwives training for the Midwife Teachers Diploma awarded by the Central Midwives Board. The college is at High Coombe, Kingston Hill, Surrey, and will accommodate 16 students at a time for courses lasting six months. The first course is expected to begin in January, 1950. Intending candidates, who must satisfy the requirements laid down by the Rules (Section C) framed by the Central Midwives Board under the Midwives Acts, should apply for places to Mr. G. Thomas, Secretary, Midwife Teachers Training College Council, 72, Great Peter Street, London, S.W.1. Selected applicants will be required to give the council of the college a written undertaking that if they qualify they will serve as midwife teachers within the National Health Service for at least two years. The Minister of Health hopes that hospital boards and committees will encourage midwives likely to make good teachers to take advantage of these facilities. Suitable intending students should not be dissuaded from taking the course on the grounds that the hospital in which they are employed has no prospective vacancies for midwife teachers, since the scheme is intended to benefit the midwifery services as a whole.

RISK OF NEUROLOGICAL COMPLICATIONS IN PERNICIOUS ANAEMIA TREATED WITH FOLIC ACID

BY

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AND

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The identification and synthesis of pteroyl-γ-glutamic acid (folic acid*) by Angier *et al.* (1945, 1946) led to the early recognition of its quite unexpected and remarkable effects in the treatment of pernicious anaemia (Moore *et al.*, 1945) and of nutritional macrocytic anaemia (Spies *et al.*, 1945).

The results in pernicious anaemia were very striking at first, as many workers reported, including ourselves (Wilkinson, Israëls, and Fletcher, 1946). It soon became evident, however, that even when the blood counts were restored to normal levels relapses were occurring, and signs of involvement of the peripheral nerves, such as paraesthesiae and numbness in the limbs, and of the spinal cord tracts, like ataxia and loss of vibration sense, might not be improved or might become worse.

Even more important, however, was the fact that some patients who had not previously shown signs of nervous system disturbances developed such signs, often very acutely, after being treated with folic acid for variable periods. Since 1946 several groups of workers have become aware of this disquieting fact, and in consequence the widespread enthusiasm for folic acid, at least for the treatment of pernicious anaemia, has been rapidly waning.

TABLE I.—*Summary of Cases Reported as Developing Neurological Changes*

Authors	No. of Patients Treated	No. Developing Neurological Symptoms and Signs	Neurological Symptoms Made Worse
Schwartz, Armstrong (1947)	69	4	—
Heinle, Welch (1947); Heinle *et al.* (1947); Spies *et al.* (1948)	47	3	—
Spies, Stone (1947); Vilter, Vilter, Spies (1947); Vilter, Vilter, Hawkins (1947)	38	28	7 (out of 19)
Ross, Belding, Paegel (1948)	22	9	4
Israëls, Wilkinson (1949); Wilkinson (1948, 1949)	20	13	3
Keil (1947)	19	1	—
Davidson, Girdwood (1947)	16	1	—
Hall Watkins (1947); Hall, Watkins, Hargraves (1946)	14	"A significant proportion"	—
Wagley (1948)	10	8	—
Castle, Berk (quoted Wagley, 1948)	10	1	—
Bethell, Sturgis (1948); Bethell *et al.* (1946)	6	1	—
Jacobson *et al.* (1948)	—	2	—

In Table I we have included all the available references to the development of subacute combined degeneration of the spinal cord in patients with pernicious anaemia, or the worsening of neurological symptoms already present, after treatment with pteroyl-γ-glutamic acid.

From a careful study of the various reports it was obvious that many of them referred to the same groups of cases, since some authors have published their results and cases in several journals, often without cross-references, and in some reports no details are given at all, other than

*In this paper folic acid is used synonymously with and refers to the synthetic pteroyl-γ-glutamic acid.

the bare statement relating to the occurrence of neurological complications in a group of patients. We have therefore inserted in the table the latest figures of each group of workers, omitting their earlier ones in other papers.

These reports suggest that, though folic acid treatment for patients with pernicious anaemia will lead in most cases to a satisfactory remission of the anaemia temporarily, there is a risk that damage to the spinal-cord tracts may continue and may show itself even after the blood counts have been restored to normal (Wilkinson, 1948, 1949). It is quite clear that pteroyl-γ-glutamic acid will not control disease of the central nervous system already present when treatment is begun. Previous estimates of the risk of the development of nervous system disturbance have varied in the larger groups from three out of 47 (Heinle and Welch, 1947; Heinle *et al.*, 1947) to 28 out of 38 (Spies *et al.*, 1948). Except for the later groups few of these patients have been observed for more than 12 months; most of them were under control for shorter periods. The pernicious-anaemia-posterolateral-sclerosis syndrome is notoriously slow to develop, so in order to obtain a better estimate of the risk involved in the use of synthetic pteroylglutamic acid we have since early in 1946 been observing closely a group of 20 pernicious-anaemia patients, either in relapse or having had no previous treatment, only three of whom had initial evidence of subacute combined degeneration of the spinal cord. We treated these patients with pteroyl-γ-glutamic acid alone until signs and symptoms of central nervous system disease appeared, or until there were signs of exacerbation of the disease initially present, and we have observed them all at frequent intervals: our intermediate results have been reported elsewhere (Wilkinson, Israëls, and Fletcher, 1946; Wilkinson, 1948, 1949). Our results after three years' observation of the group show that at the time of writing (May, 1949) only four of these patients are still well on folic acid treatment.

Illustrative Cases

The patients are divided into four groups: (1) Four patients with signs of disease of the nervous system developing acutely during treatment; (2) nine patients with signs of disease of the nervous system developing gradually during treatment; (3) three patients who had disease of the nervous system initially; and (4) four patients who had not developed signs of disease of the nervous system at the time of writing.

Details of groups 1, 2, and 3 are given in Table II, and of group 4 in Table III. Illustrative case records of patients in groups 1, 2, and 3 are given below. The patients in group 4 had the usual features of pernicious anaemia without neurological disturbance.

TABLE III.—*Group 4 Cases (No Signs of Disease of Nervous System)*

Case No.	Age	Sex	Initial Count		Period of Treatment (Months)	Final Count		Continuing Dose Folic Acid (mg. daily)
			R.B.C. 10^6/c.mm.	Hb g./100 ml.		R.B.C. 10^6/c.mm.	Hb g./100 ml.	
17	76	M	1·42	5·0	25	4·96	14·8	10
18	71	F	2·08	5·5	27	4·70	14·6	2·5
19	67	F	1·54	4·4	29	4·00	11·3	10
20	47	M	1·47	5·0	37	4·98	14·8	10

Group 1: Case 1

A married woman aged 63 was seen for the first time in May, 1946, complaining of dyspnoea, loss of weight, and increasing fatigue. She was pale and jaundiced, there was no enlargement of liver or spleen, and little abnormal was found in the heart and lungs. The arm and leg reflexes were normally active, plantar responses were flexor, and there were no subjective or objective sensory changes. A blood count showed: red cells, 1,640,000 per c.mm.; haemoglobin, 5.2 g. per 100 ml. The bone marrow was typically megaloblastic. There was complete achlorhydria gastrica.

She was admitted to hospital and given synthetic pteroyl-γ-glutamic acid, 20 mg. daily, by mouth. Her clinical condition and the anaemia improved steadily. After four weeks her red cells numbered 3,460,000 per c.mm. and the haemoglobin was 10.7 g. per 100 ml. Folic acid was reduced to 10 mg. daily, and after five months' treatment, when the haemoglobin was 14.2 g. per 100 ml., it was reduced to 2.5 mg. daily. This seemed to be too little, because after nine months the haemoglobin had fallen to 13.4 g. per 100 ml. and the red cells to 4,200,000 per c.mm. It was after nine months that she complained that her legs felt "as if she had wet stockings on," and examination showed that vibration sense was absent. The dose of folic acid was raised to 5 mg. daily. The nervous symptoms remained stationary until the 15th month, when within seven days she lost the use of her legs and became unable to walk. The blood count was then: red cells, 4,070,000 per c.mm.; haemoglobin, 11.8 g. per 100 ml.

Examination showed exaggerated reflexes, extensor plantar responses, absent vibration sense, and much-impaired position sense.

Folic acid treatment was immediately discontinued, and she was given liver extract intramuscularly and 30 g. of desiccated stomach daily by mouth. After one month her walking was improved, but the right plantar response was still extensor; after two months she was ambulant again and both plantar responses were flexor; after eight months all symptoms had disappeared and she has remained well since.

TABLE II.—*Cases in Groups 1, 2, and 3. Patients Developing Neurological Changes on Folic Acid Treatment*

Case No.	Age	Sex	Initial Count		Initial Neurolog. Signs	Period of Treatment (Months)	Final Hb Level g./100 ml.	Increasing Anaemia Present	Final Dose of Folic Acid mg. Daily	Signs of Disease of Nervous System							Recovery on Liver or Stomach (Months)
			R B.C. 10^6/c.mm.	Hb g./100 ml.						Plantar Response	Knee-jerks	Ankle-jerks	Romberg's Sign	Paraesthesia Numbness	Vibration Sense	Joint Sense	
1	63	F	1·64	5·2	None	15	11·8	Yes	5	Extensor	+++	+++	+	+++	Absent	Impaired	2
2	63	F	1·15	3·6	None	18	14·6	No	5	Flexor	Dim.	—	+++	++	,,	Absent	8
3	50	F	0·95	3·3	None	22½	10·5	Yes	30	,,	Dim.	—	+++	++	,,	Impaired	3
4	73	M	2·20	7·4	None	3	10·5	No	20	Doubtful	+++	+++	—	+++		Absent	—
5	51	F	1·82	5·8	None	12	11·8	Yes	20	Flexor	+	++	—	++	,,	,,	3
6	55	M	2·15	7·7	None	13	12 5	Yes	10	,,	Dim.	Dim.	—	+++	,,	Impaired	3
7	45	M	1·53	5 0	*a*	5	11·8	Yes	15	,,	+	+	—	+++	+	Present	3
8	56	F	2·06	7·2	None	6	13·8	No	20	,,	+	+	+	+++	+	,,	6
9	58	M	1·21	3·9	None	23	8·8	Yes	10	,,	++	++	+	+++	Absent	Impaired	2
10	48	M	1·80	6·6	*b*	3	13·0	No	20	,,	++	++	+++	+++	,,	Absent	2
11	64	F	2·00	8·3	None	19	13·3	No	40	,,	Dim.	Dim.	—	+++	,,	,,	2
12	56	F	1·31	5·2	Paraesth.	2	8·6	No	20	Extensor	++	—	—	+++	,,	,,	—
13	31	F	1·31	4·8	None	35	14·4	No	10	Flexor	++	++	++	++	,,	Impaired	3
14	53	M	2·92	7·2	*c*	2½	14·6	No	20	,,	++	++	+	+++	,,	Absent	1
15	46	M	3·83	13·3	*d*	1	13·3	No	20	,,	++	++	—	+++	,,	Present	1
16	70	F	2·13	8·8	Paraesth.	6	13·8	No	20	,,	+	—	—	+++	,,	Impaired	3

a = Stiffness and deformity of right arm present for 29 years, due to extrapyramidal lesion. *b* = Joint sense absent. *c* = Numbness of legs, absent vibration sense. *d* = Numbness and stiffness of fingers. Note: All patients had megaloblastic marrows initially and achlorhydria.

Group 2: Case 9

A joiner aged 58 was diagnosed as having pernicious anaemia some years before, but he had neglected treatment and when first seen his red cells had fallen to 1,210,000 per c.mm. and his haemoglobin to 3.9 g. per 100 ml. He was very pale and dyspnoeic, and had oedema of the feet and scrotum. The heart was not enlarged, but there was a generalized systolic murmur and occasional extrasystoles. His liver and spleen were slightly enlarged. In the central nervous system reflexes were normally active and plantar responses flexor; in spite of the oedema, sensation in the legs was normally appreciated. The bone marrow was megaloblastic, and achlorhydria was confirmed. He was given folic acid parenterally—at first 150 mg. intravenously, followed by 15 mg. intramuscularly on alternate days for 10 days; he was then given 20 mg. daily by mouth. He made a good recovery, and after two months, when his red cells numbered 4,200,000 per c.mm. and the haemoglobin was 11.6 g. per 100 ml., the folic acid was reduced to 10 mg. daily. After 4½ months' treatment the haemoglobin was 12.7 g. per 100 ml., and the dose was reduced to 5 mg. daily, and at nine months it was further reduced to 1.75 mg. daily, when his red cells numbered 5,060,000 per c.mm. and the haemoglobin was 13.8 g. per 100 ml. This latter dose was too small for maintenance: at 12 months the haemoglobin was only 13 g. per 100 ml., so the dose was raised to 2.5 mg. daily, and one month later to 10 mg. daily, when his red cells numbered 3,880,000 per c.mm. and the haemoglobin was 11.3 g. per 100 ml. At 14 months the red cells numbered 5,020,000 per c.mm. and the haemoglobin was 14.2 g. per 100 ml.; however, the first sign of disease of the nervous system appeared at this time—the patient complained of aching legs, but examination of the central nervous system revealed no objective abnormality.

The patient kept reasonably well until the 23rd month, when the blood count relapsed (red cells 2,930,000 per c.mm. and haemoglobin 8.8 g. per 100 ml.) and he had numbness and paraesthesiae in the arms and legs, and felt as if he were walking on cotton-wool.

Examination showed that his reflexes were still brisk and his plantar responses flexor, but vibration sense was now absent up to the knees and joint sense was impaired; Romberg's sign was present. He was given 30 g. of desiccated stomach daily, and he improved rapidly. Two months later his red cells were 4,920,000 per c.mm. and the haemoglobin was 13 g. per 100 ml. The only remaining neurological symptoms were slight paraesthesiae in the fingers.

Group 3: Case 15

A male cotton-spinner aged 46 had noticed that for the past five months his hands and fingers were becoming increasingly numb so that he could no longer handle the cotton threads; he also had some stiffness of the legs. He was not pale, and was well nourished; all tendon reflexes were brisk and equal on the two sides; the left plantar response was flexor, but the right was doubtful; cranial nerves were normal; vibration and joint sense were definitely present and normal. Other systems were normal. The blood count showed: red cells, 3,830,000 per c.mm.; haemoglobin, 13.3 g. per 100 ml. There was a definite megaloblastic marrow and an achlorhydria. The cerebrospinal fluid was not under pressure, proteins were 45 mg. per 100 ml., and there was slight globulin opalescence.

He was given folic acid, 20 mg. daily, but the blood count did not improve; after four weeks the red cells numbered 3,880,000 per c.mm. and the haemoglobin was still 13.3 g. per 100 ml.

Five weeks after starting folic acid treatment he complained that the numbness and paraesthesiae in the hands were much worse. Examination showed that vibration sense had now disappeared, but the reflexes were as before. Treatment with folic acid was therefore replaced with 30 g. of desiccated stomach. After one month of this he was much better, and his red cells numbered 4,740,000 per c.mm. and the haemoglobin was 14.2 g. per 100 ml. Ten months later he was back at work, but it was 16 months before all paraesthesiae had ceased and vibration sense had returned.

Discussion

We are not concerned here with the haematological and clinical effects of folic acid, which have been fully discussed elsewhere (Wilkinson, 1948, 1949), but only with its influence on the development of peripheral neuritis and subacute combined degeneration of the spinal cord in pernicious anaemia.

The accompanying Chart gives a graphic representation of the length of time during which treatment with pteroyl-γ-glutamic acid (folic acid) was given before the appearance

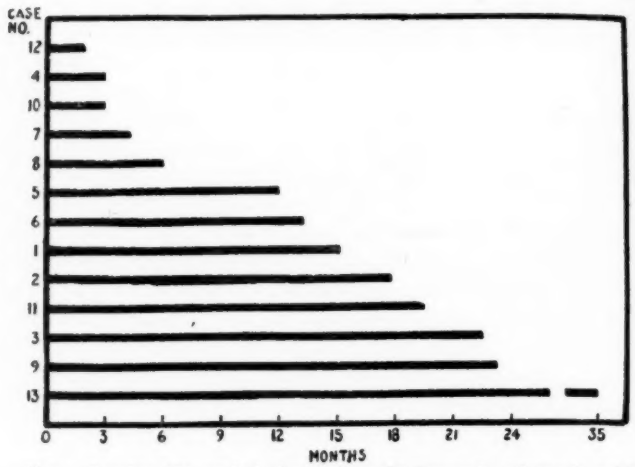

Chart showing the period that folic acid (pteroyl-γ-glutamic acid) treatment was given before the appearance of neurological signs made a change necessary.

of symptoms of subacute combined degeneration of the spinal cord. It is seen that the time of onset of neurological signs and symptoms of central nervous system disease is spread over a three-year period; apparently there is no time of special danger, nor is there any time after which the patient can be considered safe.

The relevant information about these patients in Table II shows, disappointingly, that none of these observations make it possible to predict the likely onset of neurological complications. Of the four patients in group 1 the periods of treatment varied from 3 to 22½ months. Of the 13 patients in groups 1 and 2 six showed a falling haemoglobin level, and seven did not, while the haemoglobin levels varied from 8.8 to 14.6 g. per 100 ml.

The results therefore show that in a three-year period 80% of the patients who were being treated with folic acid had developed or had increased signs and symptoms of nervous system disease that could be quite quickly controlled in the early stages by treatment with intramuscular liver extracts or oral desiccated stomach preparations. Thus there is obviously serious risk that patients with pernicious anaemia treated solely with folic acid may develop signs of disturbance of the central nervous system, or that any initial cord involvement or peripheral neuritis may get worse.

Nevertheless, four patients have been well maintained on folic acid alone for up to three years without any apparent disadvantage. Is there, therefore, any place for folic acid in the treatment of pernicious anaemia? The facts reported here and by others emphasize in a new way the dissociation between the haematological and neurological parts of the pernicious anaemia syndrome. It is well known that the neurological syndrome can occur in the absence of anaemia. We can now see that a minority (20%) of patients with pernicious anaemia do not develop (at least after three years) disease of the nervous system when an agent (folic

acid) that corrects only the haematological disorder is used for treatment. But there seems to be no way of picking out this fortunate minority; certainly, initial absence of signs of nervous disease is no help. Consequently, it is our considered opinion that folic acid ordinarily should not be used in the treatment of pernicious anaemia, and never in subacute combined degeneration of the spinal cord. Very rarely it may be of temporary use in the treatment of a pernicious-anaemia patient who cannot take stomach preparations and is hypersensitive to parenteral liver extracts, until the patient has been desensitized, although oral liver extracts will usually get over this problem.

While the acute neurological syndrome described here and by others is rarely seen in the natural course of postero-lateral sclerosis with pernicious anaemia, nevertheless its apparent direct association with folic acid therapy raises the possibility of a folic-acid/anti-folic-acid mechanism in the maintenance of the integrity of the central nervous system; but we have as yet no definite evidence that folic acid causes organic lesions in the cord. At any rate, the action of folic acid on the neurological system is not the same as that of the liver principle, although its initial effect on the bone marrow and blood appears to be very similar. We have also yet to see a patient with pernicious anaemia who would not respond to haemopoietically active liver extracts but would respond to folic acid; it turns out that such patients always have some other disease, such as idiopathic steatorrhoea.

Therefore, whatever the circumstances, folic acid, if used at all, should be replaced by liver or stomach as soon as possible, and those patients having folic acid should be seen at frequent and regular intervals so that the onset of neurological complications can be corrected without delay.

It has been our view that, while folic acid must not be used in the treatment of the pernicious anaemia syndrome on account of the risk of producing postero-lateral sclerosis in patients otherwise free from neurological symptoms, its use was allowable in the treatment of other megaloblastic anaemias that do not develop cord lesions, like those occurring in sprue, steatorrhoea, and pregnancy. This must now be reconsidered carefully in view of the recent observation by Meyer (1948) that combined system disease occurred in a patient with macrocytic anaemia secondary to ulcerative colitis of long standing. After treatment with folic acid for 18 months he quite quickly developed ataxia, loss of vibration and position sense in the legs, and positive pyramidal signs. These neurological complications appeared when the patient's haemoglobin level was normal and his weight satisfactory. Davidson and Girdwood (1948) have also reported the onset of neurological signs in two patients with idiopathic steatorrhoea who were having folic acid treatment; the signs in these patients could have been due to peripheral neuritis, and no pyramidal lesion was detected. It is only reasonable to point out that this neurological complication of non-Addisonian types of anaemia is extremely rare.

Summary

A group of 20 patients with pernicious anaemia, either untreated cases or relapsed cases, have been treated with synthetic pteroyl-γ-glutamic acid (folic acid) alone; three had initial signs of subacute combined degeneration of the spinal cord.

In a three-year period 13 patients without initial neurological involvement developed signs and symptoms of subacute combined degeneration of the spinal cord which were relieved by changing the treatment to liver extracts or desiccated stomach preparations.

Three patients with initial neurological symptoms and signs became worse neurologically after folic acid therapy.

There was no specially dangerous point during folic acid treatment, for the times at which nervous-system complications became serious were evenly distributed over three years.

Neither the presence or absence of increasing anaemia nor the haemoglobin levels could be used to predict the likelihood of neurological complications.

Four patients are quite well after two to three years on doses of 5–20 mg. of folic acid daily, given orally.

The use of folic acid for the treatment of pernicious anaemia should be strictly limited to the rare cases of special difficulty.

Folic acid should never be given to patients with subacute combined degeneration of the spinal cord.

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Delivering the chairman's address at the meeting of the Dental Board on May 11, Dr. Fish said that for the past year there had been a record decline in the total number of names on the *Dentists Register*. The figure was below that reached in 1939. About 90% of the dentists in active general practice in England and Wales, and a higher percentage of those in Scotland, were working in the National Health Service. When faced with more patients than he could comfortably treat, a dentist must do one of three things—add to his hours of work, refuse to see some patients, or speed up his work. He might even do all three. Probably most were now working longer hours than those recommended by the Spens Committee. Whether that added to their efficiency or to their enjoyment of life must remain an open question. Most dentists were in the dilemma that they must either refuse patients in need or hurry through the treatment of each in an effort to extend their services to all. The question was not whether they would condescend to work the Service but whether as individuals and as a profession they had the capacity for self-discipline, the ethical training, and the philosophy to rise to those heights of altruism which the Service demanded of them. For many there was no possibility of choice. The Service was their only means of livelihood. The success of the Service and the future of dentistry depended upon their willingness and capacity to sacrifice what might be legitimate personal gain for the salvation of their self-respect and the honour of their calling. Referring to the formation of dental companies under the influence of laymen, he said he did not think that any member of the profession should accept service under such conditions without the most careful consideration of what they implied.

AUREOMYCIN: A NEW TREATMENT FOR SYPHILIS?

BY

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Aureomycin, a yellowish crystalline substance prepared from *Streptomyces aureofaciens*, is dispensed in capsules containing 250 mg. of aureomycin hydrochloride and is administered by mouth. It has already been shown to be effective in rickettsial diseases such as Rocky Mountain fever (Ross *et al.*, 1948), in atypical pneumonia, in brucellosis (Spink *et al.*, 1948), and in certain eye conditions and urinary infections, as those due to *Aerobacter aerogenes* and *Streptococcus faecalis*, for which some consider that aureomycin is to be preferred to streptomycin and the sulphonamide group of drugs. It has considerable application to venereal disease. Finland *et al.* (1948) used it in 66 cases of gonorrhoea, with 49 definite and 11 doubtful successes and six failures Improvement was noted in two cases of penicillin- and sulphonamide-resistant non-specific urethritis. It is also of value in granuloma inguinale (Greenblatt *et al.*, 1948). In addition, Wright *et al.* (1948a, 1948b) have shown that it is effective in the virus disease of lymphogranuloma venereum.

The following case reports indicate that this antibiotic also has a powerful action upon the organism of syphilis. The patients were Bantu negroes with early syphilis, in Southern Rhodesia, and in all of them *Treponema pallidum* was demonstrable before treatment. As the supplies of aureomycin available were scanty and the prospects of an adequate follow-up problematical, it was considered expedient to treat a number with small, probably subcurative doses and to observe the effect by subsequent dark-field examination.

Case Reports

Case 1 (Seronegative Primary Syphilis).—A native from Portuguese East Africa had a typical indurated penile primary chancre with firm rubbery non-tender inguinal glands. The Wassermann and Kahn blood reactions were negative, but a dark-field examination of material taken from the sore showed *T. pallidum*. On May 18, 1949, he was given three 250-mg. capsules of aureomycin (Lederle) at intervals during 24 hours. Twenty-four hours after the starting of treatment the dark field still showed *T. pallidum*, although with greater difficulty. At 48 and 72 hours, however, none were recovered: at this stage he was given a course of penicillin.

Case 2 (Seropositive Primary Syphilis).—A male negro from Portuguese East Africa presented an indurated fraenal ulcer with a developing left inguinal bubo. Clinically the case resembled a mixed infection of syphilis and chancroid, and, although the dimelcos and Frei skin tests were negative, Ducrey's bacillus was seen in smears from the sore. The dark field, however, showed *T. pallidum*, and the Wassermann and Kahn blood reactions were positive. He was given five capsules of aureomycin orally over 29 hours (total 1,250 mg.). Twenty-four hours after the onset of treatment *T. pallidum* was still isolated by dark field, but dark-field examinations performed daily for the next 16 days were all negative. By the end of this time the sore had healed and the bubo resolved without additional treatment.

Case 3 (Seropositive Primary Syphilis).—A male negro from Portuguese East Africa was admitted with a paraphimosis and an indurated fraenal sore with typical syphilitic inguinal adenitis. *T. pallidum* was seen without difficulty in the dark field, and the Wassermann and Kahn blood reactions were positive. He was given three capsules of aureomycin daily for two days (total 1,500 mg.), and although the sore was still unhealed after 17 days no *T. pallidum* was observed at daily dark-field examinations during this time. A week later the sore had healed and the patient was discharged for surveillance.

Case 4 (Secondary Syphilis).—A Nyasaland negro attended with multiple perianal and peno-scrotal condylomata lata with enlarged non-painful inguinal glands. The dark field from the condylomata showed an abundance of *T. pallidum*, and the Wassermann and Kahn blood reactions were strongly positive. He was given one capsule of aureomycin orally three times a day for two days (total 1,500 mg.). The response was dramatic, and the lesions had healed completely by the fourth day. Daily dark-field examinations had been repeatedly negative.

Case 5 (Secondary Syphilis).—A female native of Southern Rhodesia was admitted with an indurated primary sore of the left labium majus, which was also oedematous. Secondary vulvar mucous patches and vulvar and perianal condylomata lata were also present, the whole being secondarily infected and evil-smelling. *T. pallidum* was seen without difficulty, and the Wassermann and Kahn blood reactions were markedly positive. She was given one capsule of aureomycin three times a day for two days (total 1,500 mg.), response being immediate. The daily dark-field examinations were negative for the ensuing four days, by which time the lesions had healed. She was kept under observation for a further three days; she was then discharged from hospital with instructions to attend as an outpatient. She returned, however, on the twelfth day with a relapse of the condylomata, and was then treated with penicillin.

Case 6 (Secondary Syphilis).—A female native of Southern Rhodesia was admitted with perivulvar and perianal condylomata lata, from which *T. pallidum* was obtained without difficulty. The blood Wassermann reaction was positive. She was given four capsules of aureomycin orally (total 1,000 mg.), and the lesions healed rapidly. Daily dark-field examinations were negative for the ensuing three days, and on the fourth the lesions had healed.

Case 7 (Secondary Syphilis).—A male South Rhodesian negro had a preputial sore from which *T. pallidum* was recovered and secondary condylomata lata on the scrotum. The blood Wassermann reaction was strongly positive. He was given three capsules of aureomycin orally on the first day and two on the second (total 1,250 mg.). Healing was rapid, being complete in four days, during which time daily dark-field examinations were negative.

Case 8 (Seronegative Primary Syphilis).—A male native of Southern Rhodesia had a giant fraenal chancre with typical syphilitic glands. The dark-field examination for *T. pallidum* was positive and the blood Wassermann reaction was negative. He was given six capsules of aureomycin orally in 48 hours (total 1,500 mg.). Daily dark-field examinations were negative during the next three days and the sore had nearly healed by the fourteenth day.

Case 9 (Secondary Syphilis).—A male South Rhodesian negro had an ulcerated prepuce with massive condylomata lata of the scrotum and inner thighs. *T. pallidum* was found on dark-field examination, and the blood Wassermann reaction was positive. He was given six capsules of aureomycin orally (1,500 mg.) over 48 hours. Although the dark field showed scanty *T. pallidum* at 24 hours, none was observed at two and three days. At two weeks, information was available to the effect that the condylomata had not disappeared but were quite dry.

Summary and Conclusions

Nine negro patients with early syphilis (two with seronegative primary syphilis, two with seropositive primary syphilis, and five with secondary syphilis) have been treated orally with aureomycin in doses of 750–1,500 mg. over 24–48 hours.

In six no *T. pallidum* was recovered at 24 hours, but in three the dark fields were positive at that time. In all, however, the dark fields were negative at 48 hours, and remained so daily for the periods over which observation was possible (3–17 days). Circumstances unfortunately prevented further observation.

Healing was most pronounced in the lesions of secondary syphilis, and in these the response was as dramatic as that obtained with penicillin, perhaps even more so. One case is known to have relapsed.

It is very unlikely that the small doses given were curative, although 250 mg. given four times a day for a week might well be.

It is concluded that aureomycin has a marked action upon human syphilis and opens up a new field of therapeutic investigation.

The above work was carried out during the course of a venereal-disease investigation conducted for the Health Department of the Government of Southern Rhodesia, to whom acknowledgments are due. I would also like to thank Dr A. J. V. Wilkins, Medical Officer of Health, Salisbury, Mr. Stephen Kennedy, and the staff of the lazaretto of that city for so freely offering facilities, and Mr. E. A. Boulter for his technical assistance.

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COMMON DIFFICULTIES IN INFANT FEEDING*

BY

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One of the effects of civilization is the fact that a large proportion of human infants have become parasites on the cow. The extent to which American infants rely on cow's milk for nutriment was recently shown by Bain (1948), who states that in the North-Eastern region of the United States only 23% of all babies are fully breast-fed on discharge from maternity units. In this country the Population Investigation Committee (1948) revealed interesting geographical variations in the incidence of breast-feeding. While 24% of women in Southport were fully breast-feeding their infants at two months, 85% in Lewisham were: the corresponding figures for Cornwall and West Sussex were 45% and 80% respectively.

According to the Ministry of Health Report (1944) 80% of all babies born in hospitals in this country are discharged fully breast-fed, but 95% of those born at home are fully breast-fed at the same age. Read (1949) claims that 98% of 516 women treated by him with Waller's methods were fully breast-feeding their infants on discharge, and Waller (1946) found that 79.3% of 300 consecutive primiparae in a South London district were fully breast-feeding their babies at the age of 6 months. Kao (1948) found that 95% of 26,647 Chinese out-patients were fully breast-feeding their infants.

The most probable explanation of these wide variations lies in differences in the technique of management of lactation. The figures suggest that in some places difficulties in breast-feeding are either prevented or successfully overcome, while in others they lead to artificial feeding. Artificial feeding itself leads to other difficulties, which in turn are preventable.

The theme of this paper is the suggestion that a high proportion of the difficulties in infant feeding are man-made and preventable, and that the cause of them lies in the medical attendant rather than in an inherent defect in mother or baby.

*Read in opening a discussion in the Section of Child Health at the Annual Meeting of the British Medical Association, Harrogate, 1949.

Difficulties in Mutual Adaptation of Mother and Baby

The difficulties which arise in the mutual adaptation of mother and child in the first few days are basically due to the nature of the child, but birth injury may lead to irritability or inertia in the baby and so to difficulties in adaptation. The irritable baby (who has not had a birth injury) who screams when brought to the breast, or who approaches the breast normally and then immediately withdraws his head and kicks and cries, is made worse by attempts to discipline him.

Rigid rules about the duration and frequency of feeds which do not allow adaptation to the needs of the individual, or attempts to force the child to take food when he does not want it, serve only to increase the difficulty. The baby particularly resents efforts to hold his head or to keep the limbs at his side with the aid of tight clothes or a blanket. The irritability is still further aggravated by the unnatural system, which still has its advocates, of keeping the baby in a nursery away from his mother, of not allowing her to cuddle her baby, and of bringing the baby to the mother only at that time which is considered the right one for his feed. The mother's fondling and cuddling before a feed may go a long way towards soothing him and enabling him to take his feed more easily. These difficulties of the first few days have been ably described by Middlemore (1941).

Difficulties in the Mother

Many of the difficulties in breast-feeding arise from inadequate preparation of the mother during pregnancy. Some obstetricians take little interest in the preparation of the mother for breast-feeding, and few paediatricians are given access to the mother before her baby is born. It is the paediatrician, however, who has to help the mother to surmount the difficulties in the puerperium which may result from this neglect.

Some failures of lactation are due to the mother's lack of desire to feed her baby. This may arise from ignorance of the value of breast-feeding to the baby, from a mistaken idea of the greater convenience of bottle-feeding, or from a desire for greater freedom to visit friends and places of entertainment. She may be afraid of the recurrence of a sore nipple or mastitis experienced in a previous pregnancy. She may have been told that lactation will take a great deal out of her. She may fear that breast-feeding will spoil her figure or her clothes. She may have failed to breast-feed previous children and be convinced that she will fail again. Most of these doubts and misunderstandings can be removed by discussing them with the mother during pregnancy, explaining to her the advantages of breast-feeding to the baby and to herself, and impressing her with the pleasure and satisfaction she will derive from it.

Inadequate or improper preparation of the breast during pregnancy may be another factor of importance in causing failure of lactation. Waller (1946) advocates that the mother should be taught to express a few drops of colostrum once a day for the last two or three months of pregnancy. He has shown in carefully controlled experiments

that this practice reduces the incidence of sore nipple, overdistension, and other feeding difficulties in the puerperium. Attention to a depressed nipple during pregnancy may prevent difficulties later. The Woolwich plastic nipple shield* advocated by Waller will help to elevate such a nipple. Inadequate attention to the mother's diet and failure to treat her anaemia may also be factors in causing failure of lactation.

Social factors, which are also man-made, play their part in causing failure of breast-feeding. Overcrowding, lack of privacy, the necessity of living with relations, overwork in the home due to lack of domestic help, the necessity of return to work in industry, queueing for food, all have a deleterious effect on breast-feeding. A woman who lives in lodgings is often compelled to give her baby a bottle to quieten it. The grandmother is frequently the cause of the baby being taken off the breast.

Worry

Worry is an important factor in the failure of lactation. Delay in the coming in of the milk, or ignorance of the normal time of the coming in, may worry the mother. Careless conversation between doctor and nurse about the baby is a common source of worry to her. Routine test feeds and frequent weighings almost inevitably cause the mother to worry about her ability to feed the baby. They are very apt to mislead the doctors and nurses and cause them to give complementary feeds—which go far to convince the mother that she will not have enough milk for the baby. Even though the mother is not told the result of the test feed by the nurse, she may obtain the information she wants by other means. The frequent weighings and the obvious concern of the doctor whether there is enough milk for the baby suggest to her the likelihood of a deficiency of milk. Test feeds should be used only for specific indications, when a feeding difficulty arises which suggests that the baby is not receiving enough milk, and in these circumstances they are essential.

Mothers are worried by the practice still found in some hospitals of keeping the baby in the nursery instead of at her bedside. Fields and Rose (1948) and others have shown that a mother has a much better chance of breast-feeding her baby if he is kept at her bedside night and day and not in a nursery. When the mother hears crying at the other end of the corridor she fears that it is her baby and that he is not receiving the attention he demands. She wants to cuddle, kiss, and admire her baby, for whom she has been waiting so long, but she is not allowed even to see him except at feeding time, five or six times a day. She is not given a chance to get used to handling him—to change the napkin and bath him—and she worries about her ability to manage him when she gets home.

The early discharge from hospital, necessitated by shortage of beds and nursing staff, means that she is discharged before lactation is established, and when she gets home there is often a gap of several days before the midwife or health visitor comes to help her in her difficulties. The paediatric registrar from the maternity hospital ought to be able to visit her in her home and give her the advice which she needs.

Insufficiency of Milk

By far the commonest cause of failure of lactation is insufficiency of milk. In a considerable proportion of cases, however, insufficiency is wrongly diagnosed or results from mismanagement. Those doctors and nurses who wrongly rely on scales to provide the only guide to the progress of the infant are often misled into giving complementary feeds when they are totally unnecessary. Many are ignorant of the normal rate at which milk comes in, and expect every baby to receive 2½ oz. per lb. (19.4 ml. per kg.) from the first day, whereas frequently the mother can give only the equivalent of ¼ oz. per lb. (5.7 ml. per kg.) on the fourth day, 1¾ oz. (13.5 ml. per kg.) by the seventh day, and it is not till the tenth day or later that she can give the full 2½ oz. per lb. required to give most babies a normal weight gain. Routine test feeds are done in some hospitals at every feed. They waste a great deal of the nurse's time and take her away from important nursing duties. In the home they waste the mother's time and inevitably lead to worry. I recently saw a child who had been given a test weighing at every feed every day for nine months.

Insistence on a rapid recovery of the physiological weight loss in the newborn period and on a rapid gain in weight is another potent cause of complementary feeding. Complementary feeds, if given unnecessarily, are apt to reduce the baby's desire for the breast and so to reduce the emptying of the breast, which in turn leads to failure of milk production. Some advise that a complementary feed in the newborn period should always be given by a spoon rather than a bottle because some babies may prefer a bottle to the breast. Certainly in the first week complementary feeding should be so exceptional that no nurse in a hospital unless specially trained should be allowed to give one without the permission of the paediatrician. Nurses and doctors worry unduly about the harm which will befall the baby if it does not gain weight normally in the first few days of its life. They pay too little attention to other signs of well-being in the baby—the appearance, tone, and contentment of the baby—and too much to the weight.

The commonest time for lactation to fail is about the tenth day, when the mother first returns to work in the home. The normal falling off of milk production at this time is only temporary, and if the baby is tided over this period and, if really necessary, given judicious and never excessive complementary feeds, milk production usually returns to normal in two or three days.

A common omission in the treatment of slight insufficiency of milk is failure to empty the breast by expression after feeds. The milk so expressed may be enough to make up for the deficiency in the baby, and the complete emptying of the breast stimulates it to greater milk production.

The stimulus to milk production is lost by two other bad practices: the giving of supplementary feeds, and the use of only one breast at each feed. In either case the effect is the same—incomplete emptying of the breast for long periods, with consequent decreased milk production.

Another potent cause of the failure of breast-feeding is the ready availability of artificial feeds. Much could be achieved for positive health by all the time and money which is spent by nursing, voluntary, clerical, accountant, and transport staff in supplying tins of milk. There would be much more breast-feeding of infants if artificial feeds were not so readily obtainable. I recall the story of a midwife who, when almost at term, was seen to collect twelve tins of dried milk from the clinic.

Care of the Breast

Mismanagement of overdistension of the breast is said by Waller (1946, 1947) to be one of the principal causes of the failure of lactation. Severe overdistension leads to involution of the breast and so to artificial feeding. It

*Made by Allen and Hanburys, Ltd.

causes severe pain to the mother, and this alone adversely affects the milk supply. The baby is unable to obtain the milk partly because he cannot get his jaws behind the nipple, and he will damage the nipple by sucking it. It predisposes to infection, and so to mastitis and abscess. The condition tends to be neglected in maternity units; stilboestrol is withheld until too late, and manual expression of milk to relieve the distension is often half-hearted and performed only once or twice, so that the breast is not emptied. Stilboestrol should be given promptly in cases of unduly rapid filling which causes overdistension. The dosage in the moderate case should be 5 mg. four-hourly. It is unusual to require more than three or four doses, and if the drug is continued longer than is necessary it will stop milk production entirely.

Soreness and cracking of the nipple is another difficulty in infant feeding which is usually preventable. They may be caused by the application of hardening agents to the nipple, such as spirit, and by scrubbing the nipple with a brush. Attempts to pull off dried secretions, instead of soaking them off with saline, may cause abrasions. They may be caused by leaving the baby to suck too long on the breast in the first day or two, before the milk comes in, or later by leaving the baby too long on the breast so that he sucks long after he has emptied the breast of milk. They are caused by allowing the baby to suck on a severely overdistended breast. They may be due to faulty hygiene.

A mature baby may approximate his jaws so tightly round the areola that he creates a vacuum, and if he is pulled off without first breaking the vacuum by insertion of the finger into the angle of his mouth he may damage the nipple. All too often the sore nipple is neglected and treatment is not given. If the slightest abrasion can be detected with the hand lens the baby should be taken off the affected breast and given the milk which is expressed at the ordinary feed times. Penicillin cream should be applied to the nipple, and the baby returned to the breast when in about forty-eight hours it is healed. Neglect of a sore nipple causes a great deal of pain and worry to the mother, and may lead to mastitis and abscess formation.

A breast abscess should be regarded as a disgrace in any maternity unit, and it will not be discussed here.

Difficulties in the Baby

Excessive Crying

This is usually considered to be a sign that the baby is not getting enough milk. That diagnosis is often correct, but it is more often wrong. Doctors and nurses are far too ready to diagnose insufficiency without any proof of the correctness of the diagnosis. It is a thoroughly bad practice to take the baby off the breast without confirming the diagnosis by test feeds.

Many other babies are taken off the breast on the grounds that the milk is not suiting him, is too rich, or is watery and not strong enough. These diagnoses are always wrong and should therefore never be made. I have recently seen seven babies who were put on to the bottle because it was thought that the mothers' milk looked watery.

The many other causes of excessive crying are often overlooked. One of the commonest is a rigid feeding schedule which takes no note of the needs of the individual. It is irrational to feed the baby strictly by the alarm clock. Such a method presupposes that babies are not individuals but are all alike. It presupposes that they have the same stomach-emptying time, the same degree of gastric acidity, the same temperament, and the same degree of activity. It ignores the wide changes of temperature characteristic of the British climate. It ignores the fact that babies have rest periods and periods of activity, and that activity causes hunger and thirst.

Gesell and Ilg (1937) and Aldrich and Hewitt (1947) have done many babies a great service by advocating the self-demand schedule, a rational method of infant feeding now widely practised throughout the United States. I have never yet seen any difficulties arising from it, but I have seen it solve very many feeding difficulties which arose from rigidity of method. Even if one does not go all the way in allowing complete freedom for the baby to choose its feed time, one should certainly advise the mother to use considerable elasticity in the feeding schedule and adjust it so far as she possibly can to the needs of her child. She should not waken the child for a feed. She must realize that the smaller the baby the more often it is likely to want to be fed. She should not give fluids between feeds because it is crying for food; instead she should adjust the frequency of the feeds to the baby's requirements. She should certainly be advised to feed the baby in the night in the first month or two when he wants it. Refusal to feed the baby in the night is a potent cause of excessive crying and of loss of sleep to mother and father.

Other causes of excessive crying are overclothing, neglect, loneliness, and lack of cuddling. The worst case of prickly heat which I have seen in this country was that of a baby who was brought to me on a particularly cold winter's day for excessive crying. The child was grossly overclothed and sweating profusely. Tight clothes which prevent free movements of the limbs may cause a baby to cry. Failure to change the napkin when it is wet is an obvious cause of unhappiness. A very common cause of excessive crying is loneliness and boredom.

Many mothers have the entirely erroneous belief that they will spoil a baby if they pick him up when he cries. Some have learnt a little about what psychologists call "overstimulation." They certainly will not spoil him unless they carry it out to excess. In the older baby the crying is frequently due to nothing but boredom, the baby being left outside in the pram for many hours a day, often without any toys, when he has learnt new skills—manipulation, sitting, or standing, which he is not given the opportunity of practising.

Rigidity in the duration of the feed is another cause of excessive crying. Those who advocate a fixed time on the breast for all babies presuppose that all babies suck at the same speed and that all breasts are the same. The duration of the feed is an individual matter. It is wrong to instruct all mothers to feed their babies exactly ten minutes at each breast. After the first two or three weeks most babies obtain all the milk they want in about five minutes at each breast. A very common cause of wind and colic is leaving the baby to suck too long on a breast. When the breast is empty sucking inevitably causes air swallowing.

Still another manifestation of wrongful rigidity of ideas concerns the quantity of milk the baby should take. Many doctors ascribe excessive crying and other feeding difficulties in the breast-fed baby to overfeeding. If they do a test feed and find that it is getting more than 2½ oz. per lb. (19.4 ml. per kg.) they feel that their diagnosis is confirmed. This diagnosis is almost always wrong. I have yet to see a baby whose symptoms are due to its taking too much from the breast. Not all babies are satisfied with the normal 2½ oz. per lb., and not all babies will show a satisfactory weekly gain in weight on that amount. The baby knows how much he needs.

Further Difficulties

Inadequate Weight Gain.—This is by no means always due to insufficiency of milk. It may be due to dehydration from excessive clothing. I was recently asked to see a baby who would not gain weight in spite of the fact that he was receiving an adequate supply of milk and that no organic disease could be found. The temperature of his room was about 80° F. (26.7° C.). The baby was covered with fifteen layers of clothes, including four layers of blankets. When the baby was suitably clothed the weight gain was normal. Defective weight gain may be due to a low-grade infection of the umbilicus, skin, finger, or nose. It may be due to separation from the mother in hospital and consequent loneliness and excessive crying. It is hoped that all children's units of the future will cater for the admission of the mother to the same room as her baby. Just when the baby most needs his mother, when he is ill or in pain, he is deprived of her love and cuddling, and he is left alone more than he has ever been left before.

Diarrhoea.—I have recently seen six babies who were taken off the breast on account of diarrhoea. In each case the diarrhoea was due to the mother taking certain purgatives which affect the breast milk—senna, aloes, cascara, or rhubarb. Many babies are thought to have gastro-enteritis or some form of protein or carbohydrate dyspepsia because of loose green stools which in fact are due to constipation arising from severe underfeeding. Many other babies are thought to have diarrhoea when in fact their stools are the normal loose stools of the fully breast-fed infant. Gastro-enteritis is an extremely rare cause of diarrhoea in the breast-fed baby.

Constipation.—True constipation in a fully breast-fed infant is usually due to insufficiency of milk. In a large proportion of babies brought up for this complaint, however, the motions are normal, but the mother is worried about their infrequency. It is perfectly normal for some breast-fed babies to have a motion only every four to five days. If the child is well and gaining weight normally and the motions are not hard, no treatment of any kind is necessary. I saw an infant 24 hours old who had been given an enema by the doctor for constipation.

Vomiting.—Many babies are thought to be vomiting, whereas they are merely posseting or bringing a little milk up with wind. Many babies are taken off the breast on account of vomiting. This is always a wrong thing to do, unless the vomiting is due to severe flatulence arising from insufficiency of milk. I have recently seen seven cases of congenital pyloric stenosis in which the baby had been put on to artificial foods because of vomiting, it being thought that the breast milk was "not suiting" the baby. In every case the vomiting persisted.

Flatulence.—More often than not flatulence is thought to be the cause of the baby's excessive crying and other symptoms, and very often the diagnosis is incorrect. Flatulence may, however, be caused by leaving the baby to suck on the breast too long or by insufficiency of milk. The latter diagnosis should be confirmed by test feeds.

Other Man-made Difficulties in the Breast-fed Baby.—Soreness of the mouth and thrush may be caused by cleaning the mouth of the newborn with swabs or by giving the baby a "dummy." A variety of infections may be caused by inadequate hygiene. Prematurity, with all its associated feeding difficulties, may be due to poor nutrition, obstetrical procedures, and other factors. Inertia in the newborn, leading to difficulty in breast-feeding, may be due to obstetrical procedures and to improper use of analgesics. Some babies have to be taken off the breast because of admission to a hospital which has no accommodation for a nursing mother. Others are taken off the breast on account of congenital haemolytic anaemia. Cathie (1947) showed that this was a wrong practice. Others are taken off unnecessarily because of the onset of menstruation.

Some Difficulties in the Artificially Fed Baby

Many of the difficulties which occur in the breast-fed baby also apply to the artificially fed baby. In addition certain special difficulties may occur.

Excessive flatulence and therefore colic is often due to the hole in the teat being too small. I saw a case of excessive flatulence of very recent onset in a 6-months-old baby: it was due to the fact that the mother had filled the feeding bottle with sago pudding and expected the baby to suck it out through the ordinary teat. Flatulence is also commonly due to leaving the baby to feed himself from a bottle propped up on a pillow. This causes him to swallow air for two reasons: he may create a vacuum in the bottle, and when the teat flattens in consequence he continues to suck it, so that he merely swallows air. It may also be due to the teat not being kept filled with milk, with the result that the baby swallows a mixture of air and milk.

Constipation is usually due to underfeeding. It may also be due to insufficiency of fluid or to failure to add sugar to those dried foods which do not contain added sugar, such as National Dried Milk.

Diarrhoea may be due to an infection such as gastro-enteritis or to adding sugar to a dried food which already has a high carbohydrate content. Diarrhoea must be distinguished from the loose green constipation stools already mentioned.

Vomiting may be due to improper mixing of the milk or to excessive flatulence. It is occasionally due to the hole in the teat being too large so that the baby gets the food down too quickly.

Failure to gain weight satisfactorily is usually due to underfeeding, but it may be due to infections or other organic disease. It may be due to measuring dried milk with a teaspoon which gives much less than the expected drachm (3.5 ml.), or to excessive dilution of the milk, either deliberately or because of the use of a tablespoon as a measure of ½ oz. (14 ml.) (many tablespoons measure ¾ oz. (21 ml.) or more). It may be due to following the instructions on the packet instead of adjusting the quantity of food to the needs of the individual. Very exceptionally it may be due to overfeeding. Overfeeding on the bottle can occur, but it is rare. It is certainly not nearly as common as Gibbens (1949) suggests. He is quoted as making the assertion that nine-tenths of all diseases in infancy and childhood are due to overfeeding. In fact most babies know when they have had enough, and overfeeding is an extremely rare finding in infants. It must be remembered that some babies require more than the calculated requirement to make them gain weight normally.

A common practice in the face of these feeding difficulties is to change the feeds in order to try to find a particular food which will "suit" the baby. This is a bad practice. The modern dried foods are so good that any of them if given properly and in reasonable quantity will cause most babies to thrive. Some cause other than unsuitability of a particular brand of milk should always be sought for the child's symptoms.

Difficulties in Weaning

Most of these are due to mismanagement. The commonest difficulties arise from trying to force the baby to

take food which he does not want. A determined baby will not tolerate such forcing. Attempts to give solids before the baby can chew may lead to vomiting. When a baby refuses the food offered it is a good plan to offer him a breast-feed first and then try the solid food after the breast-feed. When he gets used to it the breast-feed is stopped. The same applies to the baby who spits out the cod-liver oil and defies all efforts to give it to him. He will often take it at the end of a breast-feed without difficulty. A baby's crying and fussing during a feed is apt to be considered mere naughtiness, when in fact it is due to thirst, a soiled napkin, fatigue, or teething. It may be due to his desire to feed himself, or to a desire to use a cup instead of a bottle. Attempts to force the child to feed when he does not want it will certainly increase the difficulty. Many mothers are worried by the normal falling off of the weekly weight gain after the first six months, and so of his appetite. If the mother tries to force him to take extra food he will certainly refuse it. The more anxious and fussy the mother, the greater the difficulties which arise. The calm, patient mother who does not force the baby to take food against his will rarely has serious difficulty in feeding her baby.

Conclusions

A very large proportion of feeding difficulties are man-made. They arise from failure to prevent difficulties, from ignorance of the normal variations, and from rigidity and inelasticity in infant feeding instead of adapting the method of feeding to the needs of the individual. Other difficulties arise from the smattering of knowledge possessed by many about nutrition. Ultimately most of the difficulties in infant feeding can be ascribed to the inadequate training of doctors and nurses in the practical details of infant feeding and to the concentration in their teaching on the rare and the obscure, and on complex laboratory investigation and procedure, instead of on the really common problems which they will meet every day in general practice.

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Within the next six or eight months the Northern Ireland Tuberculosis Authority hopes to have an additional 200 beds, including 116 for children. Dame Dehra Parker, Minister of Health and Local Government, stated recently that there were 1,381 cases of tuberculosis undergoing treatment in hospital, and that the number awaiting admission was 625. She added that there were now 248 more beds available than on September 1, 1947, when the Authority assumed all functions in connexion with tuberculosis services. The use of sleeping shelters at the homes of patients in suitable circumstances was being encouraged. At present 124 shelters were in use, and approval had been given to the provision of a further 50.

ANTIHISTAMINES IN THE TREATMENT OF NAUSEA AND VOMITING OF PREGNANCY

REPORT ON A CLINICAL INVESTIGATION

BY

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Nausea and vomiting of varying degree occur so often during pregnancy as to be accepted by the patient and her attendant as normal accompaniments of that condition. In some cases, however, in the absence of adequate treatment the vomiting becomes progressive, leading eventually to a complete intolerance of all foods and liquids.

Apart from those cases in which the sickness is associated with disorders of the urinary and gastro-intestinal systems, etc., little is known regarding the origin of the nausea and vomiting of pregnancy, although most clinicians seem to accept neurosis as an aetiological factor of considerable importance. Theories have been advanced which relate the condition to endocrine upsets and dietetic deficiencies; and the usual occurrence of the sickness during the period of placental development has led to speculation on the possible part played by the placenta in producing it. The most interesting hypotheses are those which consider that vomiting of pregnancy is an allergic reaction to albumin of ovo-placental origin, and that it is caused by toxic placental products, such as peptone, guanidine, and histamine, liberated into the blood stream by the activity of the trophoblast.

The conception that histamine might be an important aetiological factor was advanced by Hofbauer in 1926, and Kapeller-Adler (1941) has revived interest in this work. She has investigated the histidine-histamine metabolism in normal and pathological pregnancies. From her results she concludes that the toxaemias of late pregnancy are probably manifestations of histamine intoxication, and that the disorders of the early months, such as morning sickness, excessive salivation, dyspepsia, and headache, may indicate a smaller degree of such intoxication.

The encouraging results obtained from the application of antihistamine drugs to the albuminuria of acute nephritis led to an attempt to determine whether or not the onset of albuminuria as a manifestation of toxaemia could be averted by the use of these drugs in pregnancy. It was soon found that patients who complained of nausea and vomiting experienced relief from their symptoms during the administration of the antihistamine drugs. The response obtained, particularly in cases in which other measures had failed, was so striking that it was decided to carry out a fuller investigation of the therapeutic potentialities of the antihistamines in this condition.

Present Investigation

This clinical investigation was carried out during the period from January 1, 1948, to March 31, 1949, when a total number of 1,816 new patients attended the antenatal clinics of the Public Health Department of the County Borough of Smethwick. In the early part of the investigation observations were made on the effect of an antihistamine drug on patients whose nausea and/or vomiting did not respond to the therapeutic measures generally employed in obstetrics. These measures consisted in advice

on diet, such as the partaking of frequent small meals with abundant carbohydrate, the elimination of fried foods from the diet, and the avoidance of items such as tea and coffee, which in some instances seemed to aggravate the vomiting. All patients were advised to try the effect of eating a biscuit or two before rising from bed. "Marmite," yeast tablets, and dilute hydrochloric acid were also used, either singly or, in certain cases, in combination. The use of the antihistamines as primary agents in treatment was advocated later as the effects of their application were noted. Furthermore, as confidence in the drug was gained—having particular regard to the fact that no untoward reactions of any kind concerning the pregnancy itself were noted—it was found possible in some cases to give a larger therapeutic dose. The drugs used in the investigation were "anthisan" and later, when it became available, "phenergan."

The dosage of anthisan originally employed was one 0.1-g. tablet morning and evening, and this dosage was gradually increased until the drug was being given as a routine five or six times daily. The majority of the cases whose symptoms were not relieved completely by anthisan treatment occurred in the early stages of the investigation, when the smaller doses were given. This gave the impression that a stronger antihistamine drug would lead to better results, and accordingly, when phenergan became available for clinical trial in the latter part of the investigation, it was used in preference to anthisan. In most of the cases a daily dose of one 0.025-g. tablet three times a day proved to be sufficient to relieve symptoms. A few cases required a heavier dose, and in one case (No. 4, below) seven tablets were given daily to control the vomiting. The efficacy of the drug is dependent on the dosage employed, and its effect seems to be cumulative. It was found that the nausea and vomiting were most often relieved within two or three days of starting the drug, and treatment was continued for a period of several weeks to avoid relapse. It was usually possible to discontinue the drug after three or four weeks, but several cases were recorded in which the nausea and vomiting recurred, necessitating a further course of treatment. The chief side-effect noted with both drugs was drowsiness, and in those cases in which the effect was marked 5 mg. of amphetamine sulphate was sufficient to counteract this. The following is a summary of the results.

Total No. of cases	1,816
No. complaining of nausea and/or vomiting	899
No. treated with antihistamine drugs	94
Results of treatment:	
Cure	78 (83%)
Improvement	4 (4%)
Failure	12 (13%)

The use of the drug is illustrated in the following case records.

Case Reports

Case 1.—Mrs. A., aged 25, attended the antenatal clinic during the thirteenth week of her second pregnancy. She had suffered from severe morning sickness during the first pregnancy, the vomiting having continued intermittently until term was reached. She again complained of severe morning sickness, and was advised regarding her diet and the necessity for regular bowel action. When seen again four weeks later, however, she was no better and had lost 3 lb. 12 oz. (1.7 kg.) in weight. Anthisan, one 0.1-g. tablet three times daily, was prescribed for her, and after two weeks of treatment her sickness had disappeared completely. The drug was discontinued and she remained well for a period of four weeks, when vomiting of a milder degree recurred. The drug was therefore prescribed again for a period of one week, after which the vomiting ceased entirely. Apart from the vomiting the pregnancy remained normal throughout and culminated at term in the spontaneous delivery of a healthy female infant of 6 lb. 8 oz. (2.95 kg.).

Case 2.—Mrs. B., aged 38, attended the antenatal clinic in the ninth week of her seventh pregnancy. She complained of severe sickness, which occurred intermittently on several occasions each day, and stated that she had suffered thus throughout each of her pregnancies. She was given advice on general dietetic measures, and the retrodisplaced uterus was manually corrected, a Hodge pessary being inserted to maintain it in position. One week later, however, she was no better, and anthisan, one 0.1-g. tablet five times daily, was prescribed for her. At the end of one week of this treatment her sickness had disappeared entirely. The drug was then discontinued and the patient remained free of this troublesome complaint for the remainder of the pregnancy. She delivered herself spontaneously at term of a healthy male child weighing 8 lb. 10 oz. (3.9 kg.).

Case 3.—Mrs. C., aged 32, attended the antenatal clinic during the seventh week of her third pregnancy. Her two previous pregnancies had been unsuccessful, having terminated at nine weeks and eleven weeks respectively. She now stated that she had been forced to come to the clinic earlier than she had intended because of the severity of her sickness, which caused her constant trouble during the day. Phenergan was prescribed for her—one 0.025-g. tablet morning and evening. She reported a considerable improvement in her condition after four days of this treatment, and she was now sick only once each evening. The dosage was increased to three tablets a day, and this brought complete relief from her symptoms. The treatment was discontinued four weeks after her first visit. She remained well for a further three weeks, when the vomiting recurred, necessitating a further course of treatment for two weeks. Since then she has been perfectly well, and is now within four weeks of her expected date of delivery. The course of her pregnancy has otherwise been normal.

Case 4

The following case record, whilst not occurring within the above investigation, is included because of its unusual interest.

Mrs. X., aged 34, attended the gynaecological clinic in January, 1949, complaining of "one-child sterility." The only lesion discovered on examination was a cervical erosion, which was treated with the coagulating diathermy current. She was next seen during the tenth week of her second pregnancy, having been referred by her doctor because of excessive vomiting. During her first pregnancy she had required hospital treatment for hyperemesis gravidarum. The vomiting this time had started one week after the first missed period. At the beginning it had occurred two or three times daily, but had steadily become worse. Her doctor advised her on the general dietetic measures to be adopted and had tried the effect of insulin on four occasions. Despite this her condition deteriorated and eventually she was vomiting constantly by day and several times by night. During the eighth week of her pregnancy she was admitted to a Birmingham hospital as a case of hyperemesis gravidarum. She was treated for a period of 17 days along the usual lines, receiving phenobarbitone, vitamin B_1, intravenous glucose-saline, and glucose by mouth. There was some improvement in her condition with these measures, but nevertheless she was not cured completely of her vomiting and still had intense nausea when she left hospital. Her condition had been ascribed to neurosis.

Within three days of her discharge from hospital her condition relapsed and she was then admitted to St. Chad's Hospital for further treatment. Her general condition was poor and the acetone series was present in the urine in large amounts. A light diet with an abundant glucose and fluid intake was prescribed and steps were taken to rule out an associated cause of her vomiting. She was also given antihistamine drugs in the following regimen:

First Day.—Phenergan, one tablet at 6 a.m. and 10 p.m.; anthisan, one tablet at 10 a.m., 2 p.m., and 6 p.m.

Third Day.—Phenergan, one tablet at 6 a.m., 4 p.m., and 10 p.m.; anthisan, one tablet at 10 a.m., 1 p.m., and 8 p.m.

Fifth Day.—Phenergan, one tablet at 6 a.m., 12 noon, 5 p.m., and 10 p.m.; anthisan, one tablet at 10 a.m., 2 p.m., and 8 p.m.

Tenth Day.—Phenergan, two tablets at 6 a.m. and 10 p.m., and one tablet at 12 noon, 4 p.m., and 7 p.m.

The drugs were given in this manner in an effort to avoid, if possible, any feeling of drowsiness and also to make use of the minimum efficient dosage. It was found necessary to prescribe 5 mg. of amphetamine sulphate each morning when the dosage reached seven phenergan tablets per day; this counteracted the drowsiness very effectively.

By the end of the third day of treatment her condition had improved considerably, and by the fifth day all vomiting had ceased. The nausea gradually decreased and finally disappeared completely by the eighth day. She remained absolutely symptom-free until she was discharged from hospital on the nineteenth day, looking and feeling a "different woman." Thereafter the dosage was gradually decreased until a maintenance dose of three tablets a day was reached. This was discontinued after a period of three weeks; the symptoms did not recur, and at the time of writing this patient is approaching the end of her second trimester of pregnancy.

Commentary

The treatment of nausea and vomiting of pregnancy still constitutes a major problem in antenatal care. It is well recognized that spontaneous relief of symptoms may be expected by the end of the first trimester of pregnancy, and the majority of patients resign themselves to awaiting this happy outcome. Most cases, fortunately, are of mild degree and do not appear to require any special treatment. When treatment is necessary simple dietary measures usually suffice to give adequate relief. In many cases, however, despite such treatment, the annoying symptoms persist and render life miserable even if they do not constitute a potential danger to it. Such being the case, there is a need for a simple and safe method of treatment which will relieve the symptoms in those patients who do not find dietary measures sufficient.

The results of the investigation into the use of anthisan and phenergan in this condition suggest that the antihistamine drugs may offer just such a form of therapy. The majority of patients who received treatment with these drugs had already been treated along the usual lines, and, even allowing for the fact that some of these patients could have expected eventual relief without further treatment, it is difficult not to be impressed with the results obtained. At any rate, the success of this method has made it desirable to carry the investigation further with a series of patients treated in various other ways.

I am indebted to Messrs. May and Baker, Ltd., for supplies of anthisan and phenergan, and to the medical advisory staff of that firm for their advice and assistance.

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ANAEMIA OF PREGNANCY IN GLASGOW AND DISTRICT

BY

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The importance of anaemia as a complication of pregnancy seems to have diminished in recent years in many parts of the British Isles. For instance, Fullerton, Mair, and Unsworth (1944) found that in Aberdeen only 3.9% of pregnant women of the hospital class suffered from anaemia, and comparison with previous surveys in that area indicates a rapid diminution in the number of cases since 1935 (Davidson, Fullerton, and Campbell, 1935). Similar observations have been made in Edinburgh (Davidson, Donaldson, Dyar, Lindsay, and McSorley, 1942; Davidson, Donaldson, Lindsay, and Roscoe, 1944).

It is therefore surprising to find that in Glasgow anaemia is one of the commonest complications of pregnancy and among the most serious. Unfortunately no previous investigation has been made in this area, and it is therefore impossible to assess progress.

The following attempt to survey the present position on Clydeside and to discover the cause of this high incidence of anaemia is based on the investigation of 4,595 patients at the antenatal clinic of the Glasgow Royal Maternity and Women's Hospital during 1947 and the latter half of 1946.

Methods

These were the standard procedures adopted by most haematological departments. Venous blood, withdrawn without constriction, was collected in heparinized tubes from every patient attending the antenatal clinic, and all investigations were carried out within six hours. Haemoglobin estimations were made by the Sahli method, 65% on this scale being equivalent to 70% Haldane, or 10.36 g.%. The haemoglobinometers were standardized at regular intervals against blood of known oxygen capacity. Pipettes requiring no correction factor were used.

Most authors consider 70% Haldane to be the lowest normal value during pregnancy. Roscoe and Donaldson (1946) have suggested that this lower limit varies with the patient's age, and that pregnant women in younger age groups are in an anaemic state at haemoglobin levels which come within the range of normality for older women. In view of this we have classified our patients into three groups: those with haemoglobin readings falling within the range 60–65% Sahli were classified "low normals"; those with readings between 50 and 60% were considered moderately anaemic; and those with readings below 50% were regarded as severely anaemic.

Classification of Patients

We found that 17.6% of all patients were anaemic at their first visit to the antenatal clinic. In addition a further 2.5% seen early in pregnancy became anaemic at some later date in pregnancy. These latter patients were in the "low normal" group originally. This gives a high incidence of 20.1%. Owing to the natural curve of haemoglobin values occurring in pregnancy certain corrections

were necessary before we arrived at this figure: these will be discussed later. The majority were cases of moderate anaemia, but approximately 5% of all patients had a severe anaemia with haemoglobin readings below 50% Sahli (8 g.%).

Since the numbers were so great and the majority of patients appeared at the clinic for the first time just before term it was impossible to investigate each case fully. Accordingly 140 were chosen for investigation in some detail over an average period of 2.2 months, and in no case less than one month. The average duration of pregnancy at first attendance was 5.6 months, with a range of from three to eight months. All anaemic patients were treated empirically with iron, and according to their response to that treatment these 140 patients were eventually classified into three groups: (A) 112 (80%) patients who showed satisfactory progress; (B) 26 (18.6%) patients who maintained their original haemoglobin level but failed to improve; and (C) two (1.4%) patients who deteriorated.

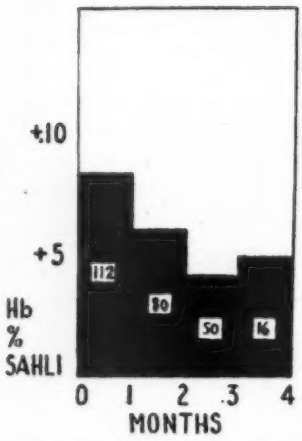

FIG. 1.—Whole of Group A. Monthly haemoglobin increments of 112 cases treated with iron as out-patients.

Group A

The results of treatment in this group are shown in Fig. 1. It will be seen that the increase in haemoglobin was greatest in the first month of treatment, and that the rate of increase diminished slightly in succeeding months. Seventy-seven of these patients were only moderately anaemic, and a graph of their monthly increments (Fig. 2) shows practically no curve. On the other hand, the remaining 35 severely anaemic patients showed a very steep curve of haemoglobin increments (Fig. 3), a rise of 10.8% being recorded in the first month, 5.4% in the second month, and 3.8% during the third month. Both curves (Figs. 2 and 3) are atypical when compared with non-pregnant cases, in which the rate of haemoglobin regeneration is said to be inversely proportional to the haemoglobin level (Whitby and Britton, 1946).

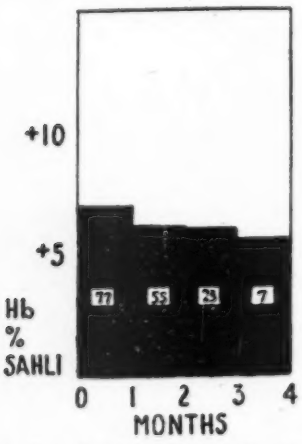

FIG. 2.—Cases of moderate anaemia in Group A. Monthly haemoglobin increments of 77 cases treated with iron as out-patients.

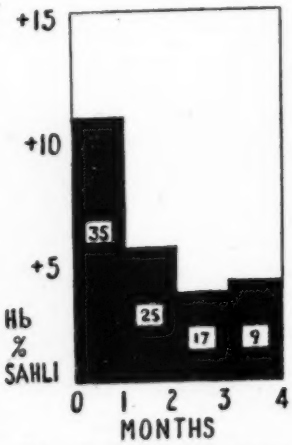

FIG. 3.—Cases of severe anaemia in Group A. Monthly haemoglobin increments of 35 cases treated with iron as out-patients.

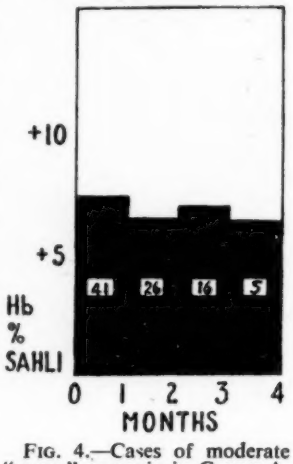

FIG. 4.—Cases of moderate "pure" anaemia in Group A. Monthly haemoglobin increments of 41 cases treated with iron as out-patients.

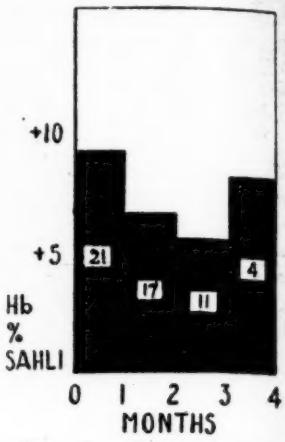

FIG. 5.—Cases of severe "pure" anaemia in Group A. Monthly haemoglobin increments of 21 cases treated with iron as out-patients.

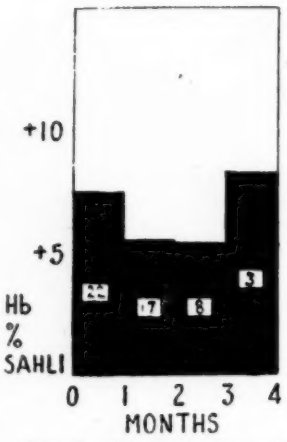

FIG. 6.—Group A cases of moderate anaemia with gastro-intestinal upset. Monthly haemoglobin increments of 22 cases treated with iron as out-patients.

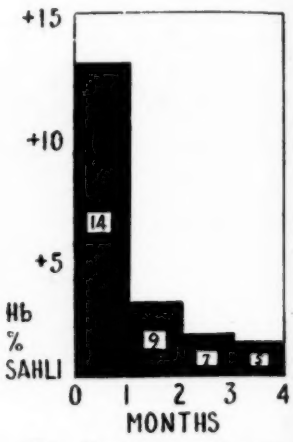

FIG. 7.—Cases of severe anaemia with gastro-intestinal upset. Monthly haemoglobin increments of 14 cases in Group A treated with iron as out-patients.

A further subdivision of these cases could be made on clinical grounds. Of the total, 62 were cases of "pure" anaemia—i.e., no other complication existed. Forty-one of these were of moderate degree, and their response is shown in Fig. 4; it was almost the same as in Fig. 2. The remaining 21 were cases of severe anaemia (Fig. 5), and these showed monthly increases which were inversely proportional to the haemoglobin level. In other words, the response of pregnant patients with uncomplicated severe iron-deficiency anaemia is the same as that shown by anaemic non-pregnant patients.

Of the remaining 50, 36 had a mild gastro-intestinal disorder: 22 of these were cases of moderate anaemia, and the curve of haemoglobin increments was similar to that of all moderate anaemias (Fig. 6); 14 were severely anaemic, the curve in their case being very steep (Fig. 7). A rise of 12.9% was recorded in the first month, but thereafter the response was only slight. It would therefore seem that a mild gastro-intestinal disturbance has little effect on the course of a moderate anaemia, but that in severe anaemia it seriously interferes with the response to treatment. Since the initial response in these cases was satisfactory it is unlikely that the gastro-intestinal disturbance

produced its effect directly by interfering with the absorption of iron. It is possible that it interfered with the utilization of iron, or it may have produced deficiencies of other haematinic factors.

The remaining 14 patients suffered from mild infections or high blood pressure, but the numbers in each group were too small to allow any further subdivision. The response to treatment did not appear to be affected. Most were cases of moderate anaemia.

While assessing the response to treatment of these 112 patients who improved it was noted that 20 showed a delayed response to iron. During the first month the increase was less than 5%. Thereafter the response was satisfactory (Fig. 8). The incidence of gastro-intestinal disorders and infections and the average duration of pregnancy were the same in this group as for the whole series. Neither these factors nor the severity of the anaemia could account for the delayed response. It is interesting to note that the response to iron therapy in this particular group of 20 patients is exactly the reverse of that seen in non-pregnant cases of anaemia. The importance of this group lies in the fact that no anaemia can be considered refractory to iron therapy unless treatment has been continued for more than one month.

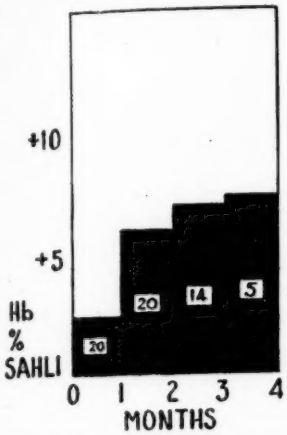

FIG. 8.—Monthly haemoglobin increments of 20 out-patients in Group A who showed a delayed response to iron therapy.

Group B

Although at first sight all of these 26 cases might be considered refractory, closer investigation suggests possible reasons for this apparent refractoriness in some. Thirteen of these patients received iron therapy for just over one month, and in view of the delayed response noted above it is not justifiable to consider them resistant to treatment. Two others developed severe infections necessitating confinement to bed for several weeks—an ample cause for their resistance (Pepper, 1927; Wintrobe, 1942; Sahli and Vaughan, 1944; Cartwright *et al.*, 1946). Another four were uncooperative, one patient improving after admission to hospital. Two patients had initial haemoglobin values of 55 and 57%. Although neither showed an increase of more than 5% in two months this was sufficient to bring them into the low normal range, and they might be incapable of further response. Only the remaining five cases of this group could be definitely regarded as resistant.

Group C

Of the two patients who deteriorated, one developed diarrhoea and vomiting in the ninth month, deteriorating thereafter. The other patient received iron for two months, during which time the haemoglobin fell 8%. She was admitted to hospital one week before term and given iron in large doses, but failed to respond although the blood picture was microcytic and hypochromic.

The first patient can scarcely be considered resistant to treatment in view of the complicating intestinal disorder. Out of the total of 140 cases of anaemia, therefore, six were definitely resistant.

Discussion

Although the survey covers a period in which many economical and social changes occurred it will be seen from Table I that there were only slight variations in the incidence of anaemia during 1946 and 1947. One out of every 20 to 25 patients attending the clinic was severely anaemic. It is interesting to note, however, that the average monthly haemoglobin readings for each of these years showed a tendency to rise in summer and fall in winter, but no correlation could be found between the incidence of severe anaemia and this seasonal variation. With the exception of a temporary rise during the Christmas season, when the numbers attending the clinic fell, this variation was quite regular, and it was also interesting to note that whereas the peak haemoglobin reading was in August, 1946, it was again reached in May, 1947, and fell rapidly thereafter.

TABLE I.—*Incidence of Anaemia*

Year	Severe Anaemias	Moderate Anaemias	Low Normals	Normals
1946	4%	17%	29%	50%
1947	4·8%	17·8%	24·5%	52·9%

From Table II it seems that age is significant only in severe anaemia. Parity is of no apparent importance.

TABLE II.—*Effect of Age and Parity*

	Age in Years	Standard Deviation	Parity	Standard Deviation
Normals	26·8	3·237	2·1	1·698
Moderate	28·3		3·5	
Average severe ..	30·7	2·812	3·6	2·810

Low Normal Series

In an attempt to elucidate the aetiology of anaemia of pregnancy a study of 85 patients whose haemoglobin values fell within the range of 60–65% was undertaken. These were followed from the fifth month of pregnancy onwards. They formed three groups.

Group I consisted of 44 patients who maintained their haemoglobin level throughout, sometimes increasing it towards term.

Group II consisted of 16 patients who showed an initial fall in haemoglobin but later improved. In none of these was the fall below 58%, and they could not be considered anaemic. Similar monthly variations in the haemoglobin values of normal patients have been noted by many authors (Meyer-Wedell, 1943; Roscoe and Donaldson, 1946; Kühnel, 1927; McGeorge, 1935; Boycott, 1936; Oberst and Plass, 1936; Thomson, Hirsheimer, Gibson, and Evans, 1938; Dieckmann, 1933). The maximum fall recorded in these patients was 4%, and these variations provide criteria for estimating the progress of any particular patient. It follows that in any patient of the "low normal" group a fall of less than 5% at any subsequent examination should not be considered significant. Doubt may be felt about the justification for this statement, since the original haemoglobin levels varied from patient to patient, and the stage of pregnancy on their first appearance at the antenatal clinic varied similarly. Statistical examination, however, showed that neither of these factors affected the validity of this conclusion.

Group III consisted of 25 patients who became anaemic. Incidentally, it was found in this group that if the haemoglobin fell below 58% Sahli the fall was progressive thereafter. Treatment was withheld until the haemoglobin fell below 55%.

A study of these three groups established certain facts relating to the onset of anaemia. Age and parity were of no significance. No difference in regard to these factors could be detected between those who maintained their haemoglobin level and those who became anaemic. Also,

since only a small percentage of "low normals" in any age group developed anaemia, pregnancy itself is not a cause of anaemia. Anaemia usually appears during the latter part of pregnancy, when foetal storage of iron is greatest. Maternal storage, however, is highest in early pregnancy, and it follows that anaemia of the later months may be related to the state of health earlier in pregnancy.

Investigation showed that 48% (12 cases) of those who became anaemic suffered from a gastro-intestinal upset in the early months, compared with 33% of those who maintained their haemoglobin level. Further, these 12 cases developed anaemia at the seventh month, whereas the remaining 13 with no such history did not become anaemic until the eighth month. Two conclusions may therefore be drawn. First, mild disorders of this nature in the early months are not a cause of anaemia later in pregnancy, although they probably hasten the onset. Secondly, the fact that the majority of those who deteriorated were healthy during the earlier months suggests that there was a relative deficiency of iron or other factors before pregnancy.

In order to test this hypothesis small groups of "low normal" patients were given dietary supplements. Twenty-five patients five months pregnant were given iron for several months. Twenty-five others received vitamin C, 150 mg. daily for one month, then 50 mg. daily till term. A similar group were given dietary advice on the type of food they should take and the best methods of preparing it. In addition, most received ½ lb. (227 g.) of liver each week for a period of not less than one month. Finally, one group received 3 dr. (11.6 g.) of "marmite" daily for one month. By that time most of them complained of nausea. All patients who deteriorated were given iron. The results of the test are shown in Table III, the control

TABLE III.—*Results of Therapy in the Low Normal Group*

Group	Improved	No Change	Deteriorated	Av. Rise in Hb After 1 Month of Iron Therapy
Control	4·7%	65·9%	29·4%	6·3%
Iron	40%	52%	8%	—
Vitamin C ..	16%	72%	12%	9%
Diet	32%	52%	16%	13%
Marmite	—	80%	20%	11%

group of 85 patients being used as a standard for comparison. From this it will be seen that iron therapy produced the best results but did not prevent the onset of anaemia in all cases. Vitamin C maintained the existing level of haemoglobin in a greater number of cases but was not so effective as iron in preventing deterioration. Its value may lie in its reducing power, thus improving the absorption of iron (McFarlane, 1936; Powell, 1944). Patients receiving special diet or marmite showed an increase in the incidence of deterioration, although not so high as in the control group. It is interesting to note the response to iron therapy of those who deteriorated in these groups. The steep rise in haemoglobin in those who had previously received special diet or marmite suggests that the rate of haemoglobin regeneration was enhanced by the diet, and that a small number of these patients had a deficiency of haematinic factors other than iron.

Summary and Conclusions

The results of the present survey show a high incidence of anaemia—namely, 17.6%—in pregnant women at their first attendance at the antenatal clinic of the Glasgow Royal Maternity and Women's Hospital. Reference to the group of low normals, however, shows that 29·4% of these patients ultimately become anaemic and a further 18.8% had readings below 60% Sahli at one time during pregnancy, though they did not develop anaemia. It is obvious that of the women attending the clinic those with haemoglobin readings below 60% at their first visit, diagnosed as anaemia, will not include the 29.4% of low normals who ultimately become anaemic, but may include the 18.8% whose haemoglobin reading is temporarily diminished. Correction for these figures gives an amended incidence of 20.1%—i.e., 20.1% of all patients seen for the first time at the clinic are or will become anaemic.

Direct comparison with other reports is difficult, but it is interesting to note that repeat surveys in other parts of Scotland show a diminished incidence of anaemia in patients attending antenatal clinics.

The reason for this high incidence on Clydeside is not apparent. The greatest emphasis has hitherto been laid on dietary iron intake. Coons (1932) estimated the daily iron requirements of the pregnant woman as 14.7 mg. Ohlson and Daum (1935) and the National Research Council, U.S.A. (1947), agree with his figure. McCance and Widdowson (1946), however, put the level as high as 20 mg. per day. Diet in the British Isles has become fairly standardized in recent years and it is therefore difficult to understand why anaemia of pregnancy should be so common in Glasgow. We have recently undertaken a dietary survey of cases of anaemia, and although to date we have examined only 16 patients the results emphasize the problem. The average daily iron intake of these patients was 16.67 mg., which is above the standard in America. It seems likely that social habits and environment may prove more important than iron intake as factors inducing anaemia in the pregnant woman.

Several points arise out of the investigation of cases of anaemia and of patients with "low normal" readings. These are best summarized as follows:

1. Thirty per cent. of all women with haemoglobin readings in the range 60–65% Sahli become anaemic if untreated.

2. Iron is the best form of preventive therapy, but an adequate intake of vitamins B and C should be maintained.

3. In these patients 58% Sahli (9.28 g. %) seems to be a critical level. Below this figure progressive anaemia develops.

4. In estimating progress a fall of less than 5% is not significant in any individual patient.

5. Pregnancy is not a cause of anaemia, and age and parity play no part in its onset.

6. Mild gastro-intestinal disorders in the early months hasten the onset of anaemia but are not responsible for it.

7. Those patients who develop anaemia are probably cases of deficient iron storage, and the defect arises before pregnancy.

8. In established cases of uncomplicated severe anaemia the response to iron therapy is similar to that obtained in non-pregnant cases. Moderate anaemias show a regular response which does not vary with the haemoglobin level.

9. When the anaemia is complicated by a gastro-intestinal disturbance there is no difference in response to treatment provided the anaemia is of moderate degree. When the anaemia is severe the response, initially good, practically ceases.

10. In 17.8% of cases the response to iron by mouth may be delayed for as long as one month.

11. An unsatisfactory response was obtained in 20% of cases, but reasons for this could be found in all but six cases, or 4.5%.

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ACUTE MASTOIDITIS IN CHILDREN TREATED WITH PENICILLIN AND SULPHADIAZINE

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It has been said that penicillin places a premium on good surgery, but is no substitute for it. While not preparing to dispute the former, the series of 17 cases recorded below would largely appear to disprove the latter.

It has been the practice at one ear-and-throat clinic at the Children's Hospital to treat as out-patients large numbers of cases of mastoiditis, early or subacute, by the extensive use of the sulphonamides and penicillin in the form of an oil emulsion, reserving for admission those cases which hitherto had appeared to demand immediate operation. While the possible risks attached to this procedure laid it open to criticism, the difficulties of obtaining adequate facilities for in-patient treatment within this or any other hospital in the vicinity were so great that it was considered a justifiable expedient.

The results, however, were so satisfactory that it was resolved to apply this method of treatment to those cases of mastoiditis which by all previous standards demanded immediate operation. The cases in this series, seen within a period of three months, are consecutive ones in which there could be no shadow of doubt of a clinical diagnosis of acute mastoiditis, and were treated by conservative methods. Cases were selected on admission and none was excluded on the grounds of its severity. Only those in which the disease had progressed so far as to produce, in addition to systemic disturbances, tenderness, swelling, oedema, and in some cases a fluctuating abscess over the mastoid process were included in the series. All showed, to a greater or lesser degree, the typical forwards and downwards displacement of the ear so well shown in the accompanying photographs. This, coupled with the appearance of the drum and meatus, left no doubt of the diagnosis nor, by the usual standards, of the necessity for operation. *X*-ray examinations were made only in the later cases in the series, and bacteriological examinations not at all. The latter is unfortunate, because it may be that the prevalent invading organisms at the time the cases were admitted were particularly penicillin- or sulphonamide-sensitive. All cases originally selected are recorded. A careful watch was kept throughout for signs of intracranial complications, but none occurred.

Treatment

On admission to hospital the following routine treatment was adopted: *Myringotomy* was performed in those cases in which the drum was intact and distended. *Intramuscular penicillin* was given in watery solution, 50,000 units six-hourly, in most cases. In the more severe cases 100,000 units were given six-hourly, and in one case 50,000 units three-hourly. *Sulphonamides* were given either four- or six-hourly, according to convenience in dosage, up to a total dose varying from 10 g. in infants to 30 g. in children of 10 years and over. No sulphonamide was given in one case in which there was acute glomerulonephritis. Sulphadiazine was used in most cases; and mist. sulphonamide co. (consisting of sulphadiazine 37%, sulphathiazole 37%, and sulphamerazine 26%) in younger children. Great attention was paid to a large fluid intake.

Local treatment consisted in careful cleaning of the meatus thrice daily and instillation of penicillin aural drops, 10,000 units per ml. As most of the children were drawn from the working-class districts of the city, one of the most important factors in the recovery of these cases was undoubtedly the excellent nursing care and attention to general health which they received.

The following is a summary of the notes of one of the most striking cases.

Case 1

Admitted April 13, 1948, aged 4 years, under the care of one of us (A. J. M.). Five weeks previously this child developed measles with otalgia and bilateral otorrhoea. The left otorrhoea ceased four weeks later, but the right otorrhoea had continued and right otalgia had been severe for the last three days. A right post-aural swelling was noticed three days before admission. It had since become worse.

On examination the child looked pale and ill. Temperature 100° F. (37.8° C.). From the right ear came a profuse muco-purulent discharge, the exact source of which was obscured by deep meatal oedema. There was considerable diffuse periostitis over the mastoid process, which was extremely tender. The pinna was pushed down, forwards, and outwards (see Fig. 1). The left drum was wrinkled but without active inflammation. Treatment consisted of systemic penicillin, mist. sulphonamide co., and penicillin aural drops.

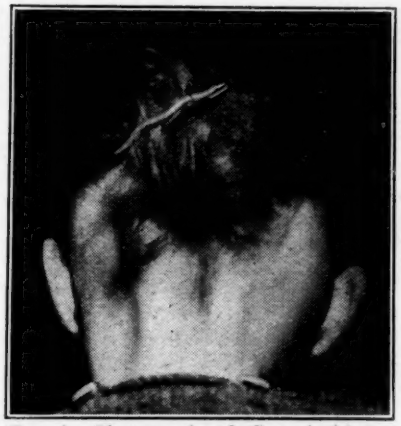

FIG. 1.—Photograph of Case 1 taken on admission.

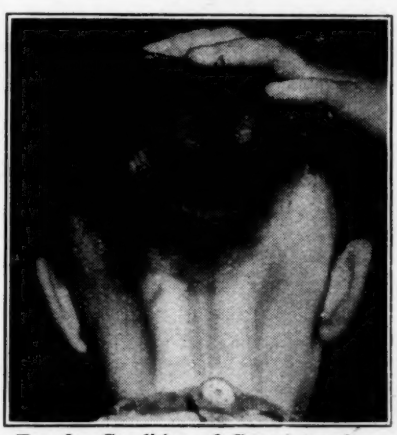

FIG. 2.—Condition of Case 1 ten days after admission.

Details of Remaining Cases

Case	Age	Duration of Symptoms	Physical Signs	Progress	Duration as In-patient	Follow-up
2	7 years	2 weeks	Right mastoid tenderness. Periostitis, especially over lateral sinus. Profuse discharge from posterior perforation	3 days after admission: otorrhoea ceased. 8 days: periostitis gone. 12 days: drum scarred; not inflamed	12 days	3 weeks after discharge: no complaints. Drum scarred; otherwise normal. Mastoid process normal. Hearing excellent. Circularized after 11 months: no further ear trouble
3	6 years	5 days	Right ear: swelling over Macewen's triangle and down post-aural groove. Copious pulsatile serous discharge from myringotomy (performed day before admission). Hearing: whispered voice at right ear	4 days after admission: otorrhoea ceased. 5 days: periostitis gone. 7 days: drum normal. Hearing: whispered voice from 5 ft. (1·5 m.) distance	7 days	6 weeks after discharge: no complaints. Right drum slightly retracted. Freely mobile. Hearing: W.V., right 10 ft. (3 m.), left 12 ft. (3·66 m.). Circularized after 9 months: no further ear trouble
4	19 months	3–4 weeks	Right ear: large post-aural swelling, pushing pinna down and out. Thick creamy discharge from posterior perforation in drum. Drum deeply injected	3 days after admission: swelling smaller but fluctuant. 8 days: swelling gone; otorrhoea ceased. 11 days: perforation healed	11 days	2 weeks after discharge: no complaints. General condition excellent. Mastoid process normal. Drum thickened but flat and white. Circularized after 10 months: no ear trouble of any sort
5	2 months	1 day	Left pinna pushed downwards and forwards by periauricular oedema. Meatus oedematous. Purulent discharge	3 days after admission: condition unchanged. 4 days: mastoidectomy decided on for next day unless much improved. 5 days: discharge ceased; meatal oedema subsiding. 11 days: postauricular and meatal swelling gone. Drum injected. 19 days: ear appears normal in all respects	19 days	10 weeks after discharge: "perfectly healthy baby"; taking feeds and sleeping well; mastoid process and zygoma normal: drum grey and normal in every respect. Circularized after 12 months: no ear trouble of any sort
6	7 years	5 days	Left drum bulging. Mastoid tenderness. Periostitis over Macewen's triangle and mastoid tip	On admission: myringotomy performed. 2 days after admission: tenderness gone. 4 days: otorrhoea ceased; swelling gone. 8 days: drum quite normal	12 days	9 weeks after discharge: "no complaints whatsoever"; mastoid processes normal; both drums quite normal and mobile. Hearing: whispered voice—right and left, 14 ft. (4·26 m.). Circularized after 11 months: had one attack of right otalgia; no trouble with left ear
7	5½ years	2 weeks	Right pinna displaced down and out. Mastoid process red and tender. Deep meatal oedema. Purulent otorrhoea	7 days after admission: periosteal thickening gone. 9 days: otorrhoea ceased. 12 days: drum thickened; otherwise normal	12 days	6 weeks after discharge: "no complaints at all. Better in every way"; mastoid process normal; drum retracted and poorly mobile; malleus prominent. Hearing: W.V. right 10 ft. (3 m.), left 12ft. (3·6 m.). Circularized after 8 months: no ear trouble
8	6 years	5 weeks	Left ear: profuse muco-purulent discharge. Periostitis over Macewen's triangle. Drum not seen owing to difficulty in examining child	6 days after admission: periostitis gone. 7 days: otorrhoea ceased. 8 days: drum white and flat	8 days	7 weeks after discharge—both drums quite normal and mobile. Mastoid processes normal. Hearing: W.V. right 12 ft. (3·6 m.); left 14 ft. (4·26 m.). Circularized after 11 months: no ear trouble of any sort
9	2½ years	4–5 weeks	Right ear: profuse purulent discharge from antero-inferior perforation. Drum deeply injected. Severe periostitis over whole of mastoid process extending above pinna along zygomatic root. No fluctuation	2 days after admission: swelling gone except for small area of thickening over centre of mastoid process. 4 days: otorrhoea ceased. 5 days: perforation healed. 9 days: mastoid process clinically normal	9 days	Circularized after 10 months: no further ear trouble
10	3½ years	1 month	Right otalgia for one month and right otorrhoea for one week. Post-aural swelling for one day. Continuous haematuria for one week. Right ear: large discoloured swelling over mastoid process, with fluctuation, maximal over Macewen's triangle; thick discharge from macerated drum. Urine: red cells and granular casts; albumin. Blood urea 31 mg./100 ml. Blood pressure 100/70. Abdomen: *nil* abnormal. Diagnosis: acute glomerular nephritis, complicating acute mastoiditis	3 days after admission: otorrhoea ceased. 5 days: swelling gone; meatus much less oedematous. Urine contains R.B.C.s and no casts. 11 days: meatus normal; drum scarred, otherwise appears normal	12 days	T. and A. 3 months later. Circularized after 10 months: no ear trouble since original attack; nephritis cleared up in 3 months
11	7 years	1 week	Previous history of severe right otalgia, 4 years and 18 months. Temperature 100·2° F. (37·9° C.). Right ear: meatal oedema, especially posteriorly; drum red and bulging; 3-point tenderness of mastoid, especially over tip	On admission: right paracentesis performed. 3 days after admission: tenderness gone. 5 days: incision healed. 7 days: drum scarred; otherwise normal	8 days	9 weeks after discharge: no complaint of ear at all; 2 head colds since discharge, but no otalgia. Right mastoid process feels normal; drum slightly retracted anteriorly, and scarred posteriorly by myringotomy incision, but freely mobile. Hearing: W.V., right 12 ft., left 12 ft. Circularized after 9 months: no further ear trouble
12	3 years	3 weeks	Left ear: fluctuant tender swelling in post-aural groove; meatal wall sagging postero-superiorly; drum macerated (mostly obscured by meatal swelling). Radiograph: "infected left mastoid"	2 days after admission: swelling much smaller. 3 days: profuse left otorrhoea started. 9 days: mastoid process normal; attack of haematuria lasting a few hours; urine contained red cells but no other abnormality; sulphadiazine stopped. 10 days: otorrhoea ceased. 11 days: meatal swelling gone; drum landmarks visible	14 days	2½ months later: no further ear trouble; drums grey and retracted
13	4 years	4 weeks	Right ear: red tender swelling over Macewen's triangle; mastoid tip tender; deep meatus oedematous; drum obscured by hard wax. Radiograph: "infected right mastoid"	4 days after admission: swelling gone except for slight periostitis over upper part of mastoid process. 8 days: mastoid process normal. 10 days: meatus normal; still not possible to remove hard wax. 15 days: drum normal	15 days	Circularized after 10 months: child well; no ear trouble of any sort
14	12 years	3 weeks	History of 3 weeks' right otorrhoea following occasional otalgia for 6 months. Right ear: severe post-aural swelling. Radiograph: "infected right mastoid." Hears whispered voice at ear	4 days after admission: swelling gone; otorrhoea ceased. 8 days: drum normal; mastoid process normal; hears whispered voice from 6 ft. (1·8 m.) both sides	9 days	1 month: right ear: drum retracted; hears whispered voice at 5 ft. (1·5 m.) right, 8 ft. (2·4 m.) left

Details of Remaining Cases (continued)

Case	Age	Duration of Symptoms	Physical Signs	Progress	Duration as In-patient	Follow-up
15	4½ months	8 days	Right ear: swelling above and in front of pinna, pushing it down and out; no fluctuation; slight discharge from myringotomy incision, performed 2 days before admission. Radiograph: "A few infected zygomatic cells on the right side"	2 days after admission: otorrhoea ceased. 5 days: swelling gone; drum appears normal. 8 days: diarrhoea. 9 days: vomiting also. 10 days: temperature and pulse rates raised; right zygoma feels warmer, and is slightly reddened and more prominent than the opposite side; right cortical mastoidectomy performed; the major part of mastoid process normal, but a chain of infected zygomatic cells found. 12 days: general condition much improved. Steady uneventful recovery	29 days	Right mastoid abscess 4 months later. Right otitis media 6 months later. Bilateral otitis media 8 months and again 11 months
16	8 years	7 weeks	Left ear: tender over lateral sinus; drum bulging postero-superiorly; purulent discharge from antero-inferior perforation	2 days: tenderness gone. 3 days: otorrhoea ceased. 10 days: drum retracted. 11 days: home	11 days	Circularized after 9 months: no further ear trouble
17	6 years	2 weeks	Right ear: profuse purulent discharge from anterior perforation. Tender mastoid tip	2 days: tenderness gone. 11 days: otorrhoea ceased	13 days	Circularized after 10 months: one attack of acute R.O.M. 4 months after discharge. Cleared up in a few days on sulphonamide and penicillin drops, given in O.P.

Progress.—April 17: Right ear dry; perforation seen to be antero-inferior; no palpable periostitis now, but ear still lower than opposite side. April 19: Perforation healed. April 23: Pinna back in normal position (Fig. 2). April 25: Could hear the whispered voice at 15 ft. (4.57 m.) both sides. Right drum normal except for the antero-inferior scar. May 5: Transferred to convalescent home.

Subsequent History.—June 17: T. and A. July 15: Both ears dry; no ear trouble since May 3. Circularized after nine months (February, 1949): No further ear trouble.

The remaining cases are described above in tabular form.

Response to treatment was immediately noticeable in the general condition of the children. This in our opinion is the most important criterion of improvement in all acute mastoid cases. If the child looks as if it were making progress it is making progress, the constitutional condition reflecting the improvement in the ear. The slightly yellowish appearance of the complexion reminiscent of the tint of a telegram envelope, easy with practice to recognize but difficult to describe, rapidly gives way to a healthy pink. This alteration in appearance is often discernible within a few hours after treatment has begun, and precedes any obvious local change. The temperature and pulse rate fall rapidly at first and then move gradually to normal. Local changes in the ear quickly follow. The redness and tenderness rapidly disappear, the oedema subsides first, and is followed by the disappearance of fluctuation as the abscess, where the infection has burst through the bone, absorbs. The ear returns to its normal position, discharge ceases, the drum regains its colour and lustre, and hearing is restored. Many of these changes are accomplished in 48 hours, and are usually complete within a week.

We consider that the end-results of this method of treatment, as shown by examination or replies to questions several months afterwards, are at least as satisfactory as if the cases had been operated on; and that after such a lapse of time any further ear trouble is more likely to be a reinfection than a "lighting-up" of the old one, and would be equally likely to occur in children upon whom mastoidectomy had been performed.

Dr. T. M. Montford, M.O.H. of Castle Donington and deputy chairman of the Leicestershire and Rutland Executive Council, was re-elected a member of the Management Committee of the Association of Executive Councils (National Health Service) at the Annual Conference held at Bournemouth recently.

ELECTROCARDIOGRAPHIC INVESTIGATION OF 264 CASES OF HYPERTENSION

BY

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Improvement in the electrocardiogram is an objective criterion to which the utmost importance has been attached in estimating the effect of treatment of essential hypertension. It has long been known that confinement to bed may in itself produce temporary improvement in the electrocardiogram. It is a matter of great interest that more comprehensive investigations carried out within recent years have revealed considerable and lasting improvement in the electrocardiograms of patients with hypertension, notably in the T-waves, as a result of dietetic treatment (Bryant and Blecha, 1947; Kempner, 1948; and others) and of sympathectomy (Canabal *et al.*, 1945; Peet and Isberg, 1946; Hammarström, 1947; and others).

There are few comparative records of electrocardiograms of untreated patients with hypertension followed up over a fairly long period. Canabal *et al.* (1945) recorded the electrocardiograms of 56 patients under 55 years of age through five years and found improvement in five cases only, all of them women under 50. They concluded that the electrocardiographic changes in hypertension tend to show that improvement occurs in few cases only, and then chiefly after acute conditions such as myocarditis and infarction. Rasmussen and Böe (1945), after six years' observation of 39 hypertensive patients, found that the electrocardiogram showed deterioration in 14 cases, while it was unchanged in 22 (of which 15 were normal) and improved in only three.

In a previous follow-up of 1,000 out-patients with hypertension (Bechgaard, 1946) with a blood pressure on the first examination of 160/100, or 180 mm. systolic or more, I had not given sufficient attention to the importance of this fact and had not followed the electrocardiograms of the individual patients with this in mind. The electrocardiograms have therefore since been revised. On the first examination 890 patients had been submitted to electrocardiography. As appears from Table I, and from a statement by Aastrup (1937), the prognosis was considerably

worse for patients with a morbid electrocardiogram on the first examination than for those with a normal one.

TABLE I

E.C.G. on First Examination	No. of Patients	Second Examination	
		Living	Dead
Normal	753	591	162
Morbid	137	54	83

Present Investigation

Of particular interest are the electrocardiograms of 264 hypertensive patients under the age of 60, all of whom were alive 11 years later and all belonging to an age at which sympathectomy may be considered. In all these cases electrocardiograms were taken at the beginning and the end of the observation period, and in several instances once or more within this period. Two-thirds of these patients were women.

The usual criteria of a myocardial lesion have been applied in interpreting the electrocardiograms. An electrocardiogram with an isolated left-axis deviation was regarded as normal. The T-waves were accurately measured. To record an improvement it was required that T1 and T2 should be at least 0.1 mV higher at the follow-up than on the first examination.

In 162 cases the electrocardiogram was normal both on the first and on the second examination; in 48 the electrocardiogram was normal on the first examination but at the follow-up showed marked deterioration, revealing signs of a myocardial lesion in all cases; and in 45 cases morbid electrocardiograms on the first examination were mainly unchanged or aggravated at the follow-up. As shown in Table II, in only nine cases had the electrocardiogram

TABLE II

	Cases
Normal E.C.G. on both examinations	162
Normal E.C.G.→ morbid on second examination	48
Morbid E.C.G. on both examinations	45
E.C.G. improved during the observation period	(9) 3
Electrocardiograms observed for from 4 to 11 years	264

improved in the course of the observation period. In three of these nine cases the T-wave in the first lead showed marked improvement, being at least 0.1 mV higher than on the first examination, and in two of them it had changed from negative to positive. In three cases a left-axis deviation had subsided, and in three some ventricular extrasystoles present on the first examination had disappeared.

It is only in the first three cases that we can say for certain that a really morbid electrocardiogram had undergone a change for the better. Hypertension persisted in all cases.

Discussion

The conclusion to be drawn from this investigation must therefore be exactly the same as that of Canabal *et al.*—namely, that definitely morbid electrocardiograms of patients with hypertension show very little tendency to improve when acute conditions, notably cardiac infarction, are left out of account, as in our cases. Improvement occurred in three patients only. This figure, though smaller, is in accord with the results of the investigations made by Canabal *et al.* and Rasmussen and Böe—particularly so when the longer observation periods of my investigations are taken into account.

Actually, these investigations give too optimistic a picture of the spontaneous improvement in the electrocardiograms, since patients who died within the observation period have not been included. The deaths among patients with a morbid electrocardiogram on the first examination were in the great majority of cases due to cardiac diseases. It is to be expected, therefore, that the proportion of spontaneous improvements would have been smaller if it had been possible to include these patients.

Lasting improvement in the electrocardiogram in hypertension, notably improvement of the T-waves, may therefore be regarded as a criterion of the efficacy of a treatment, spontaneous improvement being a comparatively rare occurrence. It may furthermore be remarked that in more than half of the patients the electrocardiogram remained normal throughout the observation period.

Great prognostic importance must also be attached to improvement in the electrocardiographic findings, since a morbid electrocardiogram must be regarded as an extremely grave prognostic sign in cases of essential hypertension. In my previous investigations into the prognosis of 1,000 hypertensive patients the mortality rates were compared with the average mortality among the Danish population, the age distribution and the length of the observation period being taken into consideration. For men with electrocardiographically demonstrable myocardial lesions the mortality was found to be 5½ times as high as the average mortality, and for women about 3 times as high, as compared with 2 and just over 1 respectively for hypertensive subjects with normal electrocardiograms.

Summary

The electrocardiograms of 264 out-patients under 60 years of age with essential hypertension have been followed for from 4 to 11 years.

Of these, 162 had normal electrocardiograms on both the first and the second examination.

Forty-five patients presented fairly unchanged morbid electrocardiograms on the two examinations.

In 48 cases a marked deterioration had occurred.

In nine cases the electrocardiogram had improved, but significantly so in three cases only.

The follow-up of 1,000 patients with hypertension disclosed a mortality rate for men with electrocardiographically demonstrable myocardial lesions 5½ times greater, and for women nearly 3 times greater, than the average mortality, against 2 and 1 respectively for hypertensive subjects without this sign.

A treatment of essential hypertension which is capable of effecting a regular and lasting improvement of the electrocardiogram must be regarded as of real value.

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The Ministry of Health states that in the removal of persons of unsound mind to mental hospitals it may be necessary in some cases to have trained staff from the hospital in attendance to accompany the patient in the ambulance or car and if necessary to assist in the removal. Requests for assistance will usually be made by duly authorized officers of local health authorities, and when hospital management committees are asked to provide trained personnel for this purpose they should do their best to comply. The smooth working of arrangements will entail the closest co-operation between officers of the hospital management committee and of the local health authority. No charge will be made to local health authorities for the services of staff provided under these arrangements.

SENSITIZATION TO DENTURE MATERIAL AS A CAUSE OF ANGULAR STOMATITIS

BY

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Cracking of the angles of the mouth may be due to many causes. These vary from mechanical maceration by dribbling resulting from ill-fitting dentures to direct infection with monilia, staphylococci, or streptococci, and to that arising in association with vitamin deficiency, particularly in ariboflavinosis. I wish to draw attention to a further cause which is not generally recognized—fissure formation due to stomatitis caused by sensitization to denture material. Of nine cases of denture stomatitis I have seen during the last four years, in seven I was consulted because of cracks at the angles of the mouth, and only after direct questioning did these patients mention painful gums. All seven cases had been treated by local applications and by various vitamins without success, some of them for several years.

Stomatitis arising from sensitization of the buccal mucous membrane to the material of which dentures are made has been recognized for many years. Prinz and Greenbaum (1939) state that "rubber sore mouth" has been known ever since Thomas W. Evans, of Paris, introduced in 1844–5 vulcanite as a base for the construction of artificial dentures. The condition is mentioned in many of the standard dermatological textbooks, such as those of Sutton and Sutton (1939), Becker and Obermayer (1940), and Andrews (1946). Lain (1932) discussed briefly the basic constituents of dental plates and stated that probably the substance mainly responsible for the stomatitis is vermilion (mercury sulphide), which, in plates that have not been sufficiently hardened, is easily liberated on to the mucous membranes. Lindsay (1928) described glossitis occurring in a patient who was shown to be sensitive to a "plastic" type of denture by patch testing. Rattner (1936) described three cases of stomatitis due to sensitization to the newer plastic materials—two of which gave positive patch tests to the dentures strapped on to the arm for 48 hours. In the third case the patch test on the arm was negative, but Goldman and Goldman (1944) have shown that it is necessary in some cases to perform the patch test on the mucous membrane to demonstrate sensitivity, and they have devised a technique for doing this.

The patch tests in the cases described below were performed by taking scrapings from the denture which was suspect and leaving these moistened scrapings in contact with the skin of the upper arm for 48 hours. There was no difficulty in reading the results. A more spectacular way is to strap the whole denture on to the skin, but my patients did not wish to be without their teeth for a period of two days.

In addition to the cracks at the corners of the mouth, the one feature common to all the patients was redness and engorgement of the whole of the mucous membrane which came into contact with the denture. The sharp line between the red inflamed denture-covered part of the hard palate and the adjoining normal uncovered part was most striking. In some cases the mucous membrane of the cheek was also affected, and one case had superficial glossitis of the part of the tongue in contact with the denture. All have been cured by changing their type of denture.

Case Reports

Case 1.—A woman aged 31, seen on January 15, 1945, complained of having had cracking at the angles of the mouth for the previous six years; this started about six months after wearing a new denture. She had failed to respond to many different preparations. There was no history of previous trouble and her general health was good. On examination the cracking of the angles of the mouth was severe and there was redness and swelling of all the mucous membrane which came into contact with her dentures. The line of demarcation on the hard palate between the engorged mucous membrane and the normal-looking posterior part of the palate was very well marked. There was no history of previous skin disease or of any relevant past illness. The dentures were made of red vulcanized rubber, and a patch test performed with scrapings was strongly positive, blisters being formed on the skin of the upper arm after contact with the scrapings for 48 hours. She was advised to stop wearing her dentures and to have a "plastic" denture after first being tested with the plastic material to be certain that she was not also sensitive to that. She was seen again on April 12, 1945, when she was completely cured. In a letter dated July 30, 1949, she stated: "I have not had any recurrence of the trouble with my mouth and gums whatsoever."

Case 2.—A woman aged 46 was seen on January 7, 1946. She gave a history of cracking of the corners of the mouth for one month, and for a week before being seen she had mild dermatitis of the circumoral region, chin, and neck. She had worn "plastic" dentures for six years, and during this time often had ulcers of her gums. There was no previous history of skin disease. She had marked cracking of the angles of the mouth, with a dry scaling dermatitis of the circumoral region, the chin, and the anterior aspect of the neck. The mucous membrane in contact with the dentures was red and engorged, and showed the sharp line of demarcation described above. A patch test to the denture was strongly positive. She was advised to change to a vulcanite denture. On July 30, 1949, she wrote saying that she "had slight recurrence of the trouble about 12 months ago which quickly responded to treatment. Apart from this I have experienced no trouble whatsoever."

Case 3.—A woman aged 44 was seen on November 22, 1946. She had had cracks at the corners of the mouth for three years continuously in spite of various forms of treatment. She first had artificial teeth in 1938, and had since noticed soreness of her gums from time to time. On examination there were deep cracks at the corners of her mouth, and the mucous membrane showed the changes seen in the other patients. The dentures were made of red vulcanite, and a patch test was strongly positive. She was fitted with "plastic" dentures, and wrote in January, 1947: "I got my new plastic teeth on December 29, and am delighted to say the cracks in my lips have quite gone and my skin is clear. My mouth has lost its soreness and I am free from ulcers."

Case 4.—A woman aged 64 was seen on August 27, 1945. On January 1, 1945, she had an irritable rash on the central part of the face, involving the forehead, nose, mouth, chin, and neck. She also had noticed cracks at the angles of the mouth during this time. She had worn dentures for about 40 years. On examination she had dermatitis of the areas mentioned above, cracks at the corners of the mouth, and marked redness and swelling of the whole of the mucous membrane in contact with the dentures. The dentures were made of black vulcanite with red vulcanite at the gums. Patch tests to the black and red vulcanite, to lipstick, and to face powder were negative. The rash and cracking persisted in spite of treatment, and in April, 1946, she was asked to discontinue wearing her dentures. She left them out only at night, but even with this the condition improved. Eventually she was fitted with a plastic denture, and wrote to me on July 29, 1949, to say that she was perfectly well, with no trace of the trouble.

Case 5.—A man aged 74 had had cracks at the corners of the mouth for about four weeks when seen on October 12, 1948. He had worn dentures for about 30 years. In addition to the cracks there was redness and swelling of the buccal

mucous membrane. A patch test to dentures was strongly positive. He was fitted with "plastic" dentures, and when seen again on March 7, 1949, the cracks had gone and the stomatitis was cured.

Case 6.—A woman aged 39 came to the out-patient department on May 14, 1949, with an infected mole. Angular stomatitis had been present for five and a half years; she had worn vulcanite dentures for about six years, and examination revealed the characteristic redness of the denture-contact area. A patch test when first tried was negative, but when repeated a delayed positive result was obtained. She was fitted with new teeth and the cracks have now healed.

Case 7.—A woman aged 58, seen on July 30, 1949, had had cracks at the angles of the mouth for many years despite many different kinds of treatment. She has worn black vulcanite dentures for 35 years. The mucous membrane showed the characteristic appearance, and a patch test to the denture was strongly positive. She has now stopped wearing dentures and the condition is much better.

Comment

These cases establish the association between denture sensitization and angular stomatitis. It is of interest that all these patients were in the habit of keeping their artificial teeth in at night, and it is reasonable to postulate that such a practice leads more readily to the establishment of a contact sensitization. In view of the numbers of dentures worn this condition is relatively uncommon, but in all the above cases the gum sensitivity was discovered only by direct questioning and examination, so that probably many cases are missed.

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ANALGESIA INHALER FOR TRICHLORETHYLENE

BY

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AND

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Of the births in England and Wales 65% are supervised by midwives (Ministry of Health, 1946). In this circumstance the problem of the relief of pain is of lively interest to nearly half a million mothers a year, but is not easy of solution. The medical practitioner can choose the anaesthetic drug and vary its concentration to meet the requirements of any particular patient at any particular moment. The midwife, however, because of her limited training in anaesthesia, is at present restricted to apparatus set automatically to deliver 50% nitrous oxide and 50% air. This apparatus of Minnitt's (1938) has proved safe, but complaints are common that it is cumbersome, and in some hands the relief from pain it affords is disappointing. This may in some degree be attributable to the fact that the female is not mechanically minded, and gas-and-air apparatus does indeed need a modicum of attention from time to time. Whether more latitude should be allowed to midwives in the way of new apparatus and drugs is a serious question. Increased relief from pain is easily achieved by raising the percentage of any anaesthetic vapour inhaled, but, quite apart from any noxious effect this may have on the foetus, it carries with it the possibility that unconsciousness and its many attendant dangers may be produced in a susceptible patient; and it must be faced that the midwife is not adequately trained to meet such a situation. With this limitation in mind work is being carried out at various centres with the object of providing better relief of pain with apparatus as safe as Minnitt's, which has rendered such useful service for the past 15 years, but more portable and less likely to present technical difficulties to midwives.

There seems to be a widespread opinion that trichlorethylene is a good analgesic agent; when carefully given it affords very considerable relief of pain without markedly impairing consciousness. In 1943 Freedman introduced a small inhaler for trichlorethylene which, under certain conditions at any fixed ambient temperature, delivers a substantially constant concentration of vapour. This inhaler proved highly convenient and successful in some hands—so much so that on the recommendation of the Anaesthetics Committee of the Royal College of Obstetricians and Gynaecologists extensive clinical trials were carried out at various hospital centres to discover whether the apparatus was suitable for use by midwives in domiciliary practice (Royal College of Obstetricians and Gynaecologists Annual Report, 1947). As a result of these trials the committee reluctantly decided that the apparatus was not satisfactory, and this view was supported by laboratory tests which showed that, even if the apparatus is kept still, the concentration of vapour in the inspired air increases by 60% for a temperature rise from 20 to 30° C. Furthermore, marked fluctuations in concentration result from filling, shaking, or tilting the inhaler.

Constant Vapour Concentration

Before discussing the design of trichlorethylene inhalers delivering a substantially constant vapour concentration, the question naturally arises whether any fixed concentration of this drug in air would improve on the relief from pain afforded by the Minnitt gas–air apparatus and yet not be dangerous.

Special trichlorethylene inhalers in which the concentration could be varied at will in accurately known steps were handed over to our colleague Dr. E. H. Seward, and his observations (1949) suggest strongly that: (*a*) A trichlorethylene concentration of 0.5% v/v in air can be inhaled intermittently during labour over long periods. (*b*) After about three hours a slight cumulative effect may become noticeable in some patients. If in these cases the concentration is now reduced to 0.35% v/v this tendency is halted. (*c*) A very large proportion of patients prefer this method of pain relief to that afforded by gas and air.

In the design of apparatus to deliver a constant concentration of vapour of an anaesthetic liquid one of the main difficulties to be overcome is the effect of variation of vapour pressure with temperature. This can be obviated either by keeping the anaesthetic liquid in a thermostat, as for example in the Oxford vaporizer, or by introducing a compensating device which, for any rise in temperature, reduces the amount of vapour leaving the vaporizing chamber. The latter method was adopted by Goodman Levy (1905) in the design of his chloroform inhaler. During the last war this department was asked to produce a dosimetric chloroform inhaler for paratroops. The resultant

E.S.O. (Epstein–Suffolk–Oxford) apparatus also was based on the second method, and utilized a compensating device which reduces the amount of air drawn through the vaporizing chamber when the temperature of the liquid rises. This apparatus stood up to vigorous tests for robustness, and gave satisfactory results both in the laboratory and clinically. Many of the principles of the E.S.O. apparatus have been incorporated in the analgesia apparatus to be described.

A Hand-operated Inhaler

In our search for a constant-concentration inhaler for trichlorethylene analgesia various experimental models were constructed with devices—some hand-operated, others automatic—to keep the inhaled vapour concentration constant despite any changes in temperature of the liquid or the surroundings. Several of these models have given encouraging results in the labour wards of this hospital for the past year. An apparatus with an automatic temperature control has an obvious advantage, but a hand-operated control is more robust and foolproof, and we incline to the belief that it will be more satisfactory in the long run. On this account we confine ourselves at the moment to a consideration of a hand-operated model, a photograph of which is shown in Fig. 1 ; the diagram (Fig. 2) is intentionally schematic.

When the patient inspires, the non-return valve (NV) opens and air enters through a variable opening (a2) into the trichlorethylene chamber and emerges mixed with vapour ; another current of room air is drawn through the permanently open by-pass of fixed size (a1) and dilutes the mixture issuing from the vaporizing chamber. If for a given size of inlet orifice (a2) and for a given temperature the concentration inspired by the patient is 0.5%, any change in temperature would alter this concentration. In order to maintain a constant concentration despite the change in temperature, compensation is effected by making the size of the air port (a2) variable. In the model illustrated the concentration can be kept constant over a temperature range from 12 to 37° C. (54 to 98° F.).

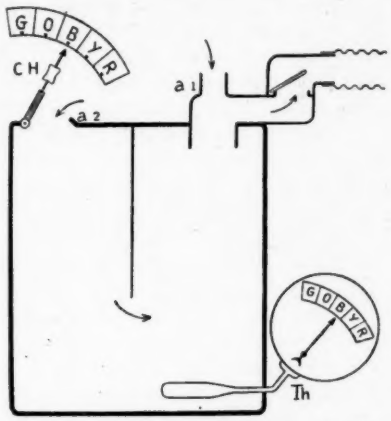

FIG. 2.—Schematic diagram of the apparatus.

On the dial of a metal thermometer (Th) are five equal sectors, each of a distinctive colour ; the centres of these sectors correspond to the following temperatures : green 15° C., orange 20° C., blue 25° C., yellow 30° C., red 35° C. The movement of the compensator handle (CH) increases or diminishes the inlet (a2) to the vaporizing chamber, and the compensator handle can be set in any of five positions coloured to correspond with the colours on the five sectors on the thermometer dial (Th).

The apparatus is so constructed that the patient breathes 0.5% trichlorethylene vapour whenever the compensator handle is set to correspond to the sector which contains the thermometer pointer at that time. If, for example, the thermometer pointer lies within the blue sector, the compensator handle has to be set to the "B" mark. It is obvious that at high temperatures, when the thermometer registers in the red field and the compensator handle is set to "R," the inlet (a2) will be at its smallest ; when the temperature is low enough to register in the green sector and CH is correspondingly set at "G," the opening (a2) will be maximal.

The heat capacity of the inhaler and heat transfer are such that the temperature of the anaesthetic liquid drops little during use in a room of reasonably constant temperature. If the temperature of the surroundings changes suddenly the internal temperature of the inhaler will follow, but only slowly. In practice, therefore, CH rarely needs to be reset. The statement that so long as CH is set to correspond to the colour indicated by the thermometer pointer the concentration of trichlorethylene vapour will be 0.5% needs but slight qualification. If the thermometer pointer is at the centre of a sector, say B, and CH is set to position B, the delivered concentration will in fact be 0.5%. If the pointer is in the right half of the sector, as shown in the diagram, the concentration will be slightly higher, and vice versa. If the pointer registers at the extreme right-hand side of a sector the concentration rises to 0.55%. However, we believe that in clinical practice such fluctuations of ± 10% in the vapour concentration are of little importance, and in speaking of "constant concentrations" possible variations within these limits are implied. It should be mentioned here that our analyses of vapour–air mixtures were carried out on samples of 2–3 litres.

The mechanism which permits delivery of a "weak" mixture (0.35%) instead of the "normal" concentration discussed so far is not shown in the diagram. It consists of a small disk which when rotated to the "weak" position opens a second air by-pass in addition to the permanent one (a1).

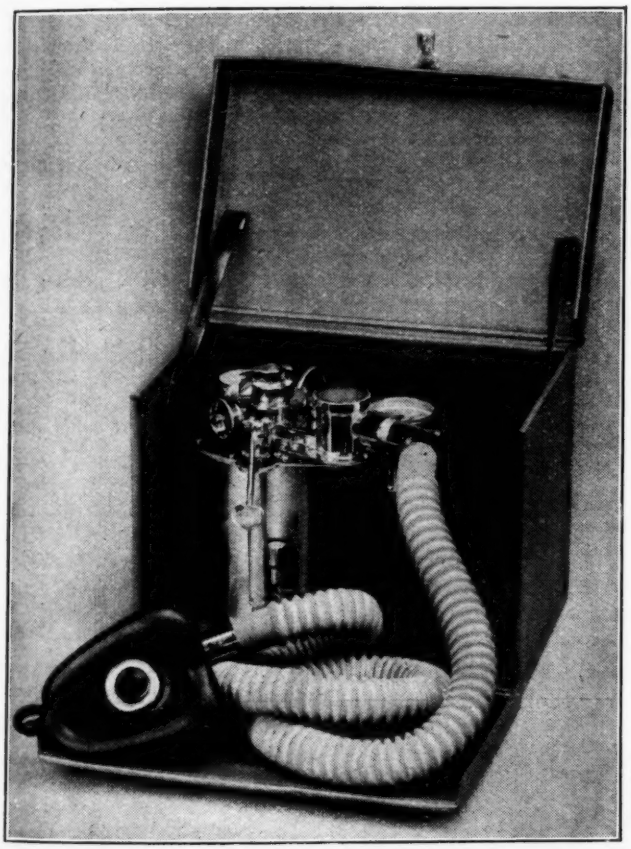

FIG. 1.—Photograph of a hand-operated model inhaler.

Besides changes in temperature other considerations must be borne in mind when designing an inhaler to deliver a fixed concentration of vapour. Unless care is taken, considerable fluctuations result from variation in the depth or rate of breathing. In the various models we have constructed the concentration remains practically constant for a range of respiratory volumes of from 6 to 15 litres per minute at rates from 12 to 30. Overfilling the vaporizing chamber with trichlorethylene is prevented by an overflow. Slow filling which occurs when the filler is at the low level of the overflow is avoided by placing the filler on top of the inhaler. Liquid does not spill to the outside during transport or if the inhaler should fall over. A glass level gauge permits an estimate of the consumption, and indicates by the "full" mark when no more liquid should be poured in. If this is overlooked the overflow comes into action almost at once. One filling is more than sufficient for a long case.

In view of the considerable cross-section of the vaporizer chamber, a special device is incorporated to give a reasonable movement of the level between "full" and "empty" corresponding to 1½ fl. oz. (42 ml.) of free liquid. The concentration of vapour remains constant whatever the position of the liquid level between the "full" and "empty" marks. Moreover, even if the level sinks out of sight, and the inhaler continues to be used, the concentration of vapour remains constant for at least three hours by virtue of trichlorethylene held in wicks. Despite the fact that the inhaler has a clearly visible liquid level—a not inconsiderable advantage—the concentration of vapour inhaled does not rise when the liquid has just been added to the inhaler, nor after the inhaler has been accidentally shaken, turned on its side, or completely inverted. These refinements would be futile if the permanent air inlet (a1) could be blocked, for the inhaler would then be dangerous in unskilled hands: accidental or intentional obstruction of this inlet has been made practically impossible. A feature of the apparatus is its low resistance to respiration compared with gas–air machines; inspiration is therefore more comfortable, and leaks due to an ill-fitting face-mask will be of less consequence in diluting the mixture reaching the patient. The apparatus is easily portable and weighs 11½ lb. (5 kg.) complete in its transit case, measuring 11 by 7 by 8 in. (27.5 by 17.5 by 20 cm.).

Some Practical Points

In using the inhaler care must be paid to three elementary points: (1) see that the liquid level lies between "empty" and "full"; (2) set the compensator handle to the colour indicated by the thermometer pointer; (3) if drowsiness or uncooperativeness should manifest itself the small disk previously referred to should be turned from "normal" to "weak," thus reducing the inhaled vapour concentration from 0.5 to 0.35% v/v.

It is unlikely that attention will have to be paid to any of these points during the course of an average labour. A very occasional glance at the thermometer pointer and control handle to confirm that the colours still coincide, and a look at the level gauge to see that it contains liquid, are all that is necessary. Movement of the non-return valve underneath its glass dome shows that the face mask fits reasonably well.

We are indebted to Imperial Chemical Industries Limited for a generous grant to meet laboratory expenses, and to our obstetrical colleagues in the United Oxford Hospitals for their willing co-operation. The apparatus in question can be obtained from the Medical and Industrial Equipment, Ltd., 12, New Cavendish Street, London, W.1, who contributed their usual enthusiasm and skill in production.

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Medical Memorandum

Recovery from Botulism

Botulism is a rare condition in this country and has a very high mortality. All of the eight affected persons succumbed in the Loch Maree outbreak of 1922. The mortality is generally considered to be at least 50%.

CASE REPORT

A woman aged 33 had an evening meal of canned tongue shortly after Christmas, 1948. The tin appeared normal, and the contents, which received no further cooking, had no unusual taste. In the early hours of the next morning she awoke with severe headache and nausea, followed by profuse vomiting. In spite of feeling very ill, she got up and went for a drive with her husband. She continued to vomit intermittently throughout the day. On returning home she found that her face was crooked and drawn to the right side. The same night the vision in the left eye was misty and swallowing had become difficult; fluids were regurgitated through the nose. For the next few days she remained in bed with mild pyrexia up to 100° F. (37.8° C.) and continued to vomit. Constipation was severe. On getting up after ten days she noted some weakness of the left leg. The difficulty in swallowing and in using the left eye persisted.

I first saw the patient on February 19, 1949, seven weeks after the onset of symptoms. The condition had not been diagnosed at the onset and consequently no antitoxin had been administered. The following signs were present: moderate dilatation of the left pupil, which was active to light but inactive on accommodation and convergence; slight left-sided ptosis; and complete paralysis of the left masseter, temporal, and pterygoid muscles. There was no facial anaesthesia and the corneal reflex was present. The left facial muscles were completely paralysed, with the exception of the orbicularis oculi, in which there was a faint contraction. Taste was normal on the left, but there was hyperacusis in the left ear. Both sides of the palate were weak. Examination of the limbs showed no abnormality other than a moderate weakness of the dorsiflexors of the left foot. The superficial and deep reflexes were normal, and there was nowhere any change in cutaneous or deep sensation.

Twenty-four hours after eating the suspected meat the patient's daughter had an attack of vomiting which continued for two days. There was no involvement of the nervous system. The patient's husband, who also ate the same meal, was unaffected.

The history of gastro-intestinal symptoms occurring a few hours after the ingestion of tinned meat and followed by paralysis of cranial nerves some hours later is characteristic of affection by the toxin of *Clostridium botulinum*. The site of action of this toxin is uncertain, but its action is probably peripheral (on the motor-nerve endings in striated and non-striated muscles) rather than central, and this theory is borne out by the absence of reflex or sensory disturbance and the ultimate complete recovery in those patients who survive the initial acute phase (Dickson and Shevky, 1923; Cowdry and Nicholson, 1924).

DAVID KENDALL, D.M., M.R.C.P.

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Reviews

THE THYROID

Pathology and Surgery of Thyroid Disease. By Joseph L. De Courcy, M.D., and Cornelius B. De Courcy, M.D. (Pp. 476; illustrated. £2 10s.) Oxford: Blackwell Scientific Publications. 1949.

Two distinguished members of the clinic at Cincinatti, Ohio, which bears their name have added yet another to the long list of recent monographs on thyroid disease and surgery. It is in most respects a thorough survey of the subject, based on experience of some 15,000 thyroid operations done over a period of almost 30 years.

The authors express a number of more or less original opinions on various topics, and these must be heard with respect. They claim, for instance, to be the originators of the view that the beneficial action of Lugol's solution in the pre-operative treatment of toxic goitre is chiefly due to a mechanical effect; the rapid storage of colloid in the vesicles of the gland produces, they say, a temporary state of pressure within the gland with a consequent oedema and limitation of the blood supply. Within a short time the effect is lost because the circulation in the gland returns to normal with adjustment to the changed state. In their survey of the physiology and pathology of the thyroid the authors emphasize the part played by the reticulo-endothelial system of the liver in the regulation of the iodine content of the blood, and discuss the value of blood-iodine estimations in diagnosis and treatment. The value of thiouracil treatment is, of course, fully discussed, and like most other unbiased observers they conclude that the chief value of these compounds is in the pre-operative preparation of the patient. The possible uses of radio-iodine are also described, but its limitations and dangers seem at present, at the De Courcy Clinic, to outweigh its virtues, The use and control of this dangerous weapon are indeed still in an experimental stage.

The difficult question of the aetiology and relationship of the two conditions known as Riedel's thyroiditis and Hashimoto's disease is fully considered, and the authors are inclined to regard them as separate clinical entities. They advance the theory that Riedel's thyroiditis begins as an inflammation outside and around the gland—a peri-thyroiditis, with fibrosis spreading into the gland from the periphery. Although the process certainly seems to affect the surrounding tissues, this explanation is unlikely to satisfy pathologists. The origin of the peri-thyroiditis is in any case left unexplained.

In this comprehensive survey the technique of thyroid surgery is given a relatively small space. It is interesting to note that the authors do not ligate the inferior thyroid artery, because they believe that the procedure "entirely destroys the blood supply of the parathyroid bodies and encourages the development of tetany." This view is contrary to the experience of most thyroid surgeons, and it is surprising that the bogy should still have any influence. The "enucleation" of thyroid adenomas from within the gland is also a procedure usually regarded as long outmoded.

A curious defect in the book is the very perfunctory account of cardiac disease in relation to thyroid surgery. The use of quinidine in the post-operative treatment of auricular fibrillation appears to be unknown to the authors, and far greater reliance is placed on the use of digitalis than would seem to be warranted by recent experience in Great Britain. A most interesting view, however, is expressed concerning the administration of desiccated thyroid gland for at least three months after thyroidectomy for toxic goitre. The authors believe that this will almost invariably prevent any recurrence of hyperthyroidism by putting the remainder of the gland entirely at rest. They even describe the history of a patient with severe toxic goitre who was cured by the administration of dried thyroid tissue without any surgical treatment at all. This subject needs further investigation.

Though not without defects, the book is interesting to read and most suggestive to any thyroid surgeon who looks beyond the mere technique of his craft. It is fully supplied with references at the end of each section.

GEOFFREY KEYNES.

VISCERAL PAIN

The Mechanism of Abdominal Pain. By V. J. Kinsella, F.R.C.S., F.R.A.C.S. (Pp. 230. £1 12s. 6d.) Sydney: Australasian Medical Publishing Company. 1948.

The author is a distinguished Sydney surgeon who for many years has devoted himself to a study of the problems of abdominal pain. He is scientifically minded, well informed, and a keen and painstaking observer; this review of his findings will be read with profit by both clinicians and physiologists. Mr. Kinsella might well have given additional space to a more detailed account of his original observations and less to a consideration of the views of the classical writers like Ross, Lennander, Head, Wilms, Mackenzie, and the rest, whose theories and concepts have in the main outlived their usefulness and now tend to cripple fresh thinking on the subject. Some of Mr. Kinsella's own theorizing—for instance, on referred pain, visceral neuroses, the "rule of superior location," and cutaneous hyperalgesia (the last partly based on an excursion into comparative anatomy)—seems to outrun our knowledge of the facts. But the rest of the book is of high value. The historical outline is illustrated by plates from the works of Vesalius, Eustachius, and Willis which put many of the figures in our modern books to shame.

The author emphasizes that the clinical study of pain is beset with difficulties and pitfalls. Pain is not just a simple sensation like muscle sense—it is accompanied by varying degrees of feeling-tone and drives. "Patients' fear and anxiety may cause the stimulation of pain or an exaggerated reaction to slight pain. The method of questioning, either the tone of the voice or the words used, may influence the patient both as to the presence and absence of pain, its degree, and its situation. A question may set the patient on edge and arouse his apprehension." A leading question may be indispensable to elicit information, but it may call forth a misleading reply. In surgical studies under local anaesthesia the patient's imagination is apt to conjure up fearsome pictures and to anticipate much suffering, and he may reply to questions in a manner calculated to safeguard himself from pain. A local anaesthetic may produce general effects: thus cocaine induces euphoria and damps down unpleasant reactions. The administration of morphine tends to suppress the slighter pains. Mr. Kinsella describes in detail his technique for operating and questioning under local anaesthesia. It may be added that once a pain is gone it is hard to recall accurately and fully; a pain "recollected in tranquillity" is merely an intellectual and emotionless simulacrum of the real experience.

The afferent innervation of the abdominal viscera is very sparse. Langley has calculated that all the afferent fibres from the viscera of the cat equal in number those in one posterior nerve root; it might be added that the fibres in all the posterior nerve roots equal in number those in the optic nerve. The visceral afferents are probably non-

medullated and finely medullated like those coming from the skin. Mr. Kinsella quotes John Hunter very aptly—"for distinct sensation, two things are necessary, namely, time and space." To arouse a sensation an adequate number of impulses must reach the brain in enough fibres over a given time to permit of "central spatial and temporal summation." In testing visceral sensibility one must therefore stimulate an adequate portion of a viscus sufficiently vigorously. There is also a great difference between the sensibility of a normal and an inflamed viscus.

These considerations may account for the discordant findings about visceral sensibility. Thus, squeezing with fingers or forceps, or stitching, cutting, or clamping the healthy appendix or caecum arouses no pain. When the appendix is inflamed squeezing it along a considerable part of its length produces pain. Wolf and Wolff similarly found that pinching the gastric mucosa or rubbing it with the blunt end of a glass rod is painless except when the stomach is inflamed and oedematous. Other observations by Kinsella on the large intestine and cervix uteri seem to prove that afferent impulses arising from certain viscera may give rise to pain—i.e., there is so-called true visceral pain. The pain fibres in the mesentery arise mainly in the gut wall itself; it is easier to produce pain by stimulating the mesentery than the gut, because in the former the fibres are concentrated in the vicinity of blood vessels while in the latter they are sparse and widely distributed. A localized stimulus applied to the gut will stimulate fewer fibres than one suitably applied to the mesentery, especially in the neighbourhood of its blood vessels. From what is known about the properties of pain fibres elsewhere it is likely that the visceral pain fibres will respond to a wide range of stimuli having in common the property of nociception. In states of inflammation the altered blood supply, oedema, and the presence of abnormal chemical products may facilitate the reaction of the pain fibres to appropriate stimuli.

Evidence is produced that the posterior pelvic peritoneum, especially over the right iliac fossa, is less sensitive than the anterior peritoneum to painful stimuli. This observation helps to explain why a retrocolic or retrocaecal inflamed appendix commonly gives rise to less pain than the normally placed organ. Mr. Kinsella discusses the mechanism of pain in peptic ulcer at some length, but no convincing conclusion emerges.

It would be well if clinicians bore in mind the limitation of physiological knowledge about pain mechanisms in general. Some account can be given of the properties of pain fibres and the course of the pain impulses from the periphery as far as the thalamus, but beyond that point our knowledge is meagre. We do not know what part of the brain is responsible for mediating either the sensation of pain or its emotional accompaniment. Stimulation of the postcentral cortex, or for that matter of any other part of the cortex, is painless. There is, however, some evidence from the results of prefrontal leucotomy that the prefrontal cortex or prefrontal-subcortical areas may be somehow concerned with the feeling-tone. Nothing is known with certainty about the way in which skin pain is normally localized with considerable accuracy. It is hardly surprising, then, that we should not know why pain of visceral origin is inaccurately localized, projected to somatic areas, or sometimes accompanied by cutaneous hyperalgesia. It is to be hoped that Mr. Kinsella and other surgeons with similar gifts and opportunities will continue to collect and record accurate studies of patients—perhaps leaving the grand generalizations to emerge gradually in their own good time in the future.

SAMSON WRIGHT.

THE FUNCTIONS OF PSYCHIATRY

Psychiatry. Its Evolution and Present Status. By William C. Menninger, M.D. (Pp. 138. 11s. 6d.) London: Geoffrey Cumberlege. 1948.

This volume of Messenger lectures founded to "share knowledge on the evolution of civilization" describes man's mental reactions and their influence on society. After early neglect psychiatry has now assumed a wide popular appeal in the U.S.A., largely because 2,000,000 young men were rejected from war service on account of personality disorders and 700,000 were discharged as unfit on this account.

The lectures begin with a brief historical survey of the rise of psychiatry and reflections on the imperfections in the teaching of this subject in relation to medicine at the present time. There are signs, however, that it is now beginning to be more effectively integrated with general medicine. The first lecture concludes with a discussion of the relationship of psychiatry with psychology, social science, anthropology, and religion.

The second lecture gives an account of psycho-analytic psychiatry under four headings—the psychosexual stages of development, the anatomy of personality in terms of the conscious and unconscious, personality physiology in terms of the id, ego, and superego (which are reasonably lucidly explained, though the concept still remains difficult to the non-technical reader), and some examples of the defence mechanisms used by the ego to maintain its equilibrium.

The third section is on psychiatry and the social order, and what can be done to improve human relationships in the post-war world. Often in mental stress a change in the environment is as important as a change in the personality, and psychiatry may show how the environment can be most wisely changed. Knowledge can only be gained by an intense study of the reaction of the individual to his environment. The importance of the family is stressed, and the increasing disturbance of family life is ominous. Disturbances in community life seem to be increasing, due to prejudices, hostilities, and such factors as housing shortages.

The prevention of mental illness is still in a backward state, though mental hygiene is making progress. The training of leaders and the instillation of the proper motivation and a spirit of service are essential to the welfare of the human race, and psychiatry can certainly contribute to these ends. These three lectures cover a vast field, but they are clear and sensible and well worthy of study by both medical and lay readers.

R. G. GORDON.

ENDOCRINOLOGY IN BRIEF

The Value of Hormones in General Practice. By W. N. Kemp, M.D. (Pp. 115. $3.) Minneapolis: Burgess. 1949.

This work describes itself as "prepared for the express purpose of assisting general practitioners in the fullest possible utilization of the many pharmaceutical hormonal preparations now available." It is not prepared in book form, but consists of cyclostyled typewritten sheets bound with wire rings. There are no illustrations (a disadvantage in a book on endocrinology), neither is there an index: there are numerous typing errors.

It is on matters of treatment that the book is least helpful. For example, Simmonds's disease (which responds well to testosterone and other hormones) has no treatment mentioned at all. Cushing's syndrome is also left devoid of therapy, and no mention is made of the adrenal, pituitary, basophil, ovarian, or thymus tumours that can cause it. The dose of thyroid for myxoedema is not specifically

mentioned except for the statement, "Forty-five grains (3 g.) of thyroid extract is the theoretical daily requirement of an adult with comp'ete athyrosis . . . smaller clinical doses are the rule," which might lead to overdosage. In connexion with pituitary dwarfism we are told, "Therapy is fairly satisfactory . . . a potent growth hormone should be administered daily . . . a growth rate considerable [*sic*] in advance of normal is desirable." Perhaps that will be true one day, but it is not true yet. The treatment of Addison's disease is quite well described, but the information that "all the thirty-nine vitamins should be present in the diet" might frighten readers that something awful would happen if they only managed to include thirty-eight. The poor balance of the book can be illustrated by there being only eight lines about the treatment of thyrotoxicosis, whereas there is more than twice that amount written about the prevention of neonatal death among sheep.

A heavy style makes the reading matter less digestible than it should be. A child, instead of being called "debilitated" or "poorly," is described as being "under a strain from concomitant biochemical or bacteriological influences"; and it would be easier to read, "Reduce the dose," instead of, "A downward revision of the dosage regimen is indicated." Though parts of the book are reasonably accurate and informative, as a whole it can hardly be recommended as a useful guide for students or practitioners of endocrinology.

RICHARD ASHER.

GUIDE TO CHEMOTHERAPY

The Sulphonamides in General Practice. By Edward D. Hoare, M.D. (Pp. 90. 5s.) London: Staples Press, 1949.

The preface to this book states that it is intended for the general practitioner, and that it attempts to indicate briefly the conditions under which sulphonamides will be valuable and the dangers which may attend their use. In addition, the relative merits of su'phonamides and penicillin are briefly discussed. The book is adequate for this purpose, and it sets out in brief, simple language most of the information which the general practitioner requires. The main principles of sulphonamide therapy are now so well established that it is not possible to be original on the subject without becoming unsound; and, if some of the statements made seem platitudinous, this is because they repeat many of the recommendations given in various previous books on this subject (although no acknowledgment is made to these sources). The book is briefer than the M.R.C. Memorandum on the sulphonamides and it contains up-to-date minor details. The arrangement of the specific infections according to whether they are more, equally, or less susceptible to sulphonamides than to penicillin is somewhat confusing. There are a few minor misprints which are disfiguring rather than important. The book can be recommended as a concise elementary guide.

F. HAWKING.

Modern Discoveries in Medical Psychology, by Dr. Clifford Allen (pp. 236; 12s. 6d.; London: Macmillan, 1949), is the second edition of a book published twe've vears ago with the object of explaining in simple language the theories and practice of medical psychology to general practitioners and students. It says much for the generation of psychiatrists, as they would now be called, of which the author is a distinguished member that such a book is much less necessary now than when the lectures on which it was founded were first delivered. Nevertheless, it is still interesting and affords us a good review of the development of the treatment of mental illness, especially psychoneurosis, which has taken place in the last half-century. Some remarks which were no doubt apposite when first written now seem out of place and might well have been omitted—for example, few contemporary neurologists seek to find a cerebral localization for hysteria.

BOOKS RECEIVED

[*Review is not precluded by notice here of books recently received*]

The British Pharmaceutical Codex, 1949. (Pp. 1,562. 63s.) London: The Pharmaceutical Press. 1949.

Handbook of Materia Medica, Toxicology, and Pharmacology. By F. R. Davison, B.A., M.Sc., Ph.D., M.B. 4th ed. (Pp. 730. 60s.) London: Henry Kimpton. 1949.

The Adrenal Gland. By F. A. Hartman, Ph.D., and K. A. Brownell, Ph.D. (Pp. 581. 60s.) London: Henry Kimpton. 1949.

Human Helminthology. By E. C. Faust, A.B., M.A., Ph.D. 3rd ed. (Pp. 744. 50s.) London: Henry Kimpton. 1949.

X-Ray Diagnosis. By G. Simon, M.D., B.Ch., F.F.R. (Pp. 207. 20s.) Cambridge: W. Heffer. 1949.

Fetal and Neonatal Death. By E. L. Potter, M.D., Ph.D., and F. L. Adair, M.D. 2nd ed. (Pp. 173. 30s.) Cambridge University Press. 1949.

A Textbook of Surgical Pathology. By C. F. W. Illingworth, C.B.E., M.D., Ch.M., F.R.C.S.Ed., and B. M. Dick, M.B., F.R.C.S.Ed. 6th ed. (Pp. 726. 45s.) London: Churchill. 1949.

A Hospital Prayer Book. 4th ed. (Pp. 47. 1s. 3d.) London: Geoffrey Cumberlege (Oxford University Press). 1949.

Science News, 13. Edited by J. L. Crammer (Pp. 160. 1s. 6d.) Harmondsworth: Penguin Books. 1949.

Lewis's Diet Charts. Revised by V. H. Mottram, 1948. (7s.) London: H. K. Lewis. 1948.

Hearing and Deafness. Edited by H. Davis, M.D. (Pp. 496. 21s.) London: Staples Press. 1947.

Mongolism (Peristatic Amentia). By M. Engler, M.D., D.P.M. (Pp. 208. 21s.) Bristol: John Wright. 1949.

An Introduction to Clinical Surgery. By C. F. M. Saint, C.B.E., M.D., M.S., F.R.C.S. 2nd ed. (Pp. 383. 45s.) London: H. K. Lewis. 1949.

A Manual of Practical Tropical Sanitation. By J. Balfour Kirk, C.M.G., M.B., Ch.B., F.R.C.P., D.P.H., D.T.M.&H. 2nd ed. (Pp. 288. 10s. 6d.) London: Baillière, Tindall and Cox. 1949.

The Filterable Viruses. By F. O. Holmes. (Supplement No. 2, Bergey's *Manual of Determinative Bacteriology.*) (20s.) London: Baillière, Tindall and Cox. 1948.

The 1949 Year Book of Medicine. Edited by P. B. Beeson, M.D., and others. (Pp. 831. 31s. 6d.) London: H. K. Lewis. Chicago: The Year Book Publishers. 1949.

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THE USES OF STREPTOMYCIN

Streptomycin is now freely obtainable in this country, and the profession as a whole is consequently in a position to make valuable use of it. At the same time there are possibilities that this expensive and by no means harmless drug may be either wasted or used in such a way as to do positive harm, and therefore its properties and indications should be kept under careful review. Streptomycin, like penicillin, is usually administered by intramuscular injection, and for most purposes, owing to its slower rate of excretion, two daily doses suffice. There is no streptomycin equivalent to procaine penicillin, with a therapeutic effect lasting 24 hours or more, though a single daily dose has sometimes been used. Unlike penicillin, its dosage has to be kept within strict limits if serious toxic effects are to be avoided: the most notable of these is on the eighth nerve, causing vertigo and disturbance of position sense and even deafness, either of which may be permanent. It is in fact a much more harmful and also a less potent chemotherapeutic agent than penicillin, and would find no place in therapeutics but for the fact that it acts on many bacteria against which penicillin is powerless.

Foremost among these is the tubercle bacillus. Much could be written about the indications for using streptomycin in tuberculosis, but they depend on two principles which can be expressed shortly. One is that the more acute the disease the greater is likely to be the effect. This probably has much to do with the fact that in the early stages of any rapid extension of the disease the bacilli are more accessible to a drug reaching the affected area through the blood: large areas of tissue have not undergone caseation and so formed a mass remote from any blood supply and therefore partially or wholly impenetrable by the drug. The strongest indications for streptomycin therapy, both on this ground and of course because they are otherwise fatal, are the miliary form of the disease and tuberculous meningitis. Rapidly extending pulmonary infiltration and ulcerative tracheo-bronchitis also call for streptomycin treatment. But when it is contemplated in a more chronic case, whether of phthisis or of the disease elsewhere, the object should be clearly defined and streptomycin administration specifically related in time and intention to other treatment, particularly surgical. Thus in advanced phthisis with extensive cavitation streptomycin may sufficiently improve the condition to make possible a thoracoplasty which could not have been undertaken before, but it should not be used until plans for this have been completed. It may be very difficult to resist the demands of patients and relatives for the new "wonder drug," but if these demands are infrequently resisted and treatment of tuberculosis with streptomycin becomes at all indiscriminate the ultimate effects will be disastrous. A warning to this effect was given in the letter of Dr. B. Couts published in a recent issue (October 22, p. 933). The fact to be borne constantly in mind is that only one chance is usually given of killing any particular tubercle bacillus with this weapon. In the majority of patients bacilli surviving at the end of a course of treatment are found to be resistant to the drug, and remain so indefinitely. This change precludes the further treatment with streptomycin both of that patient and of anyone else whom he may infect. Several instances of infection with streptomycin-resistant bacilli in contacts of treated cases have already been observed in the United States. It is unfair to the whole community to use streptomycin in patients with open tuberculosis unless for good and well-defined reasons. The decision to use it is emphatically one for the expert only.

The other uses for streptomycin are almost all included in a category which can be defined only in bacteriological terms—infections due to Gram-negative bacilli, the majority of which are little affected by penicillin. These are numerous, but some of them are either unknown in this country or uncommon. It is in tularaemia that streptomycin has had perhaps an unqualified success, but in this country we cannot exploit its usefulness either in this disease or in plague. Its disappointing effect on enteric fever is difficult to explain, but fortunately another antibiotic, chloramphenicol, has a remarkable action in typhoid.[1] Given with sulphadiazine, streptomycin will cure brucellosis, but the same effect can be achieved more simply and safely with "aureomycin." Among uncommon conditions in which streptomycin is indicated are *H. influenzae* meningitis, pneumonia due to Friedländer's bacillus, and either septicaemia or meningitis caused by such organisms as *Proteus* or *Ps. pyocyanea.*

What are the commoner conditions, other than tuberculosis, in which streptomycin may be called for? They fall into three categories—certain intestinal infections, infections of the urinary tract, and some conditions due to penicillin-resistant cocci. When streptomycin is swallowed it is neither absorbed nor destroyed, but acts as a remarkably efficient intestinal antiseptic. It undoubtedly has a beneficial effect in

[1] *British Medical Journal*, 1949, **2**, 918.

bacillary dysentery, and possibly in *Salmonella* infections other than enteric fever. Its action in infantile gastro-enteritis is a subject of controversy, possibly because there is more than one cause. It may be said that this type of use is justified only in severe cases which have been investigated as fully as possible. Most bacillary dysentery seen in this country is too mild and self-limited to deserve so costly a treatment. In ulcerative colitis streptomycin is useless. Urinary-tract infections are a different matter: owing to their frequent intractability the controlled use of streptomycin is fully justified. Whether such control is practicable outside a hospital is exceedingly doubtful. Urological investigation is first necessary to identify any underlying cause: no drug can be expected to eliminate infection in the presence of obstruction or a calculus. Examination of the urine should include determination of the sensitivity of the organisms to streptomycin and possibly to other drugs which merit trial first. It is then necessary by medication to render the urine consistently alkaline, since streptomycin is relatively inactive in an acid medium. Finally, the drug has to be given intramuscularly, and preferably—though there is singularly little information on this important point—three times a day at least. Even when such care is taken treatment with streptomycin sometimes fails, usually because some underlying cause has not been eliminated. How often, then, is success to be expected from a few intramuscular injections given optimistically to an ambulant and incompletely investigated patient? Laboratory and often other hospital facilities are also required for the investigation and treatment of the third group of infections which may be benefited by streptomycin: coccal infections normally responsive to penicillin but sometimes resistant to it. The majority are staphylococcal, though streptomycin may have a use in certain streptococcal infections. It has even been used in the treatment of penicillin-resistant bacterial endocarditis.

There are thus comparatively few conditions in which streptomycin can justifiably be used for a patient outside a hospital. Within hospital, though indications may be more numerous, the grounds for administration should be well ascertained and weighed. This warning is called for not merely because the production of streptomycin even in England means dollar expenditure, and waste is therefore the worst form of extravagance, or even only because the drug is potentially dangerous: the strongest objection to indiscriminate use is that already mentioned in connexion with tuberculosis—that unsuccessful treatment renders the organism completely resistant. The tubercle bacillus rarely acquires this property in less than about six weeks. Other bacteria, multiplying at a far greater rate, can acquire it within a few days or even overnight. The trial of streptomycin for a urinary tract infection, for instance, without proper safeguards can do a patient irreparable harm by placing him out of reach of even properly controlled treatment later. In time, no doubt, resistant bacteria of other species than the tubercle bacillus will be transmitted from person to person, and infections in untreated patients will be found totally resistant at the outset. It is even possible that the use of the drug will have to be altogether abandoned on this account in the not far distant future. Fortunately there is no other antibiotic or drug of any kind to which such high resistance can be acquired so rapidly, just as there is no other which can become an essential nutrient for an organism on which its previous action was destructive. This almost ludicrous transformation is now fully substantiated and known to occur in many species, including the tubercle bacillus. Nothing illustrates more clearly than this how fragile a weapon streptomycin is and how ill-defined is the boundary separating good from harm in its effects. Doctors possess in streptomycin something quite peculiar and not to be regarded in the same light as any other drug. It should be reserved for use only on the clearest indications and under the closest observation. Every patient treated unsuccessfully brings nearer the day when no patient will be able to be treated with streptomycin at all.

RECRUITMENT OF MEDICAL OFFICERS

The recommendations of the Spens Committees are exerting their influence far beyond the terms of reference that the committees considered. Organizations outside the Health Service competing to employ medical men—such as the universities, the armed Forces, and industrial firms—are finding that they must offer terms at least as attractive as those offered within the Health Service. Though it is doubtful whether remuneration in the Health Service fulfils the Spens recommendations in terms of the present value of money, a number of universities have found it necessary to increase the salaries paid to medical men, and the British Medical Association has made out a strong case that the armed Forces should do likewise.

Recruitment of regular medical officers to the Army, which is at an exceedingly low ebb, suggests that the financial terms offered must be improved if the Army is to be able to attract sufficient doctors, and there is reason to believe that that view is held in the War Office. The decline in recruitment can be accounted for in part as an inevitable post-war phenomenon. Research at the War Office has shown that recruitment has always fallen off after great wars, including the Crimean War, the Boer War, and the two world

wars. Though the total number of medical officers in the Army is at present sufficient for the establishment of 1.5–1.75 per thousand population at risk (which does not comprise only British troops), the number of regular medical officers is slightly below strength, and that of short-service officers is just over 60% below. The total numbers are likely to fall to about 10% below establishment by next January. The deficiency of regular officers is at present made up from National Service officers, whose work is highly praised at the War Office; they constitute about 50% of the total. This flow cannot continue in sufficient volume, for the shortage of vacancies in medical schools induces a considerable number of students to do their National Service before starting a medical course, so that they are not available to be conscripted as medical officers when they have completed it.

Specialists are particularly scarce, and the Army has recently advertised for civilian specialists at salaries that are distinctly attractive compared with those paid to medical officers, as correspondents to this *Journal* have pointed out. It is of interest to record that no specialist was appointed as a result of that advertisement, but so great is the need for them that the Army will probably continue the policy, as a temporary measure, of offering appointments to civilians. The Armed Forces Committee of the Association has considered whether it should recommend refusal of this kind of advertisement, but decided not to do so—at least for the present—in view of the Army's urgent need. It may take a different view if the Government fails to solve the Army's problem. Meanwhile many opportunities are given the regular officer to gain special experience and higher qualifications. They include study leave and work at suitable training centres, and the short-service officer is also encouraged to specialize. In all, about one-third of the regular and short-service officers are trained in this way.

The Ministry of Defence is now considering the B.M.A.'s proposals[1] for new scales of pay in the three Services. The increases proposed as a minimum range from 3s. a day in the lowest ranks to 27s. in the ranks of surgeon-captain, brigadier, and air commodore, with smaller increases in the ranks higher than those. The Government's reply is expected shortly, but the Ministry has already notified[2] the B.M.A. that remuneration of personnel in the armed Forces is decided by the Government, and that negotiations with bodies representing any section of the Forces have never been undertaken, since they would be incompatible with Service discipline. This attitude seems more calculated to frighten than to inform, for the Ministry can hardly be expecting the Association to incite its members in the Forces to sedition. No objection can be taken to the analysis of evidence and the preparation on it of a carefully reasoned case, particularly when the adoption of the Association's proposals is likely to encourage recruitment and thereby benefit the medical arms of the Services.

Though the country is in difficult circumstances economically there is no evidence that the Government intends to allow the Forces to fall below the strength vitally necessary for defence, nor would the nation allow it to do so. Further economy in medical man-power would seem to be impossible. Overlap between the three Services has already been largely eliminated; in small stations one of the Services is normally responsible for the medical care of personnel from all three, and at home the Forces co-operate with National Health Service hospitals and practitioners. The Government must therefore offer attractive terms if it is to fulfil its duty of providing adequate medical care of the armed Forces.

[1] *British Medical Journal Supplement*, April 2, p. 204.
[2] Ibid., September 17, p. 137.

ANAESTHETIC EXPLOSIONS

The risk of anaesthetic explosions should by now be well known in view of the attention it has received in recent years. In his review of the literature some four years ago Cole[1] was able to list 70 papers on this subject. They ranged from brief accounts of simple accidents in which only the doctor was damaged[2] to Greene's analysis of 230 anaesthetic fires and explosions.[3] Yet every year there are a number of anaesthetic explosions, many of which must needs be reported to the coroner. Every junior anaesthetist and probably most medical students know that the common inhalation anaesthetics used in this country—ether, cyclopropane, and ethyl chloride—are inflammable, and when mixed with oxygen explosive. Everybody knows that heat, or a spark, or a naked flame is needed to touch off an explosive mixture. It might thus appear that explosions would never occur in operating theatres, for who but the completely uninitiated would bring an explosive vapour and a source of ignition together? Yet this is what happens only too often. Two anaesthetic explosions have been reported this year[4]; many explosions in which there is no loss of life go unrecorded, though doctors, nurses, and onlookers may

[1] *Surgery*, 1945, **18**, 7.
[2] *British Medical Journal*, 1892, **2**, 1457.
[3] *Anesthesiol.*, 1941, **2**, 144.
[4] *British Medical Journal*, 1949, **1**, 117.
[5] Ibid., 1949, **1**, 244.
[6] *Amer. J. Surg.*, 1945, **69**, 89.
[7] Beckett, J., *Irish J. med. Sci.*, 1946, **6**, 656.
[8] Leech, B. C., *Canad. med. Ass. J.*, 1947, **56**, 28.
[9] *British Medical Journal*, 1936, **1**, 74.

receive injuries varying from singed hair to the loss of an eye from flying glass. The patient may be spared or may suffer fatal blast injuries of the respiratory tract.

The dangers of static electricity, which was responsible for one of these two explosions,[5] are not yet generally appreciated. These electrical charges accumulate quickly and easily on insulated objects in an operating theatre. Complacency about static electricity was produced by a once common teaching that this phenomenon occurred only in countries with a very dry climate. We know better now, and even on the dullest, dampest day a little effort in an operating theatre in smartly pulling a blanket across the top of a theatre trolley will produce a spark when a finger is held about one-sixteenth of an inch away from the metal top of the trolley. The same effect may be obtained when an anaesthetized patient wrapped in one or more blankets is lifted from a trolley to the operating table. For this reason Waugh and Bond[6] recommend that woollen blankets should never be used in the operating theatre. These static charges may be dispersed or prevented from accumulating if certain simple precautions are adopted, one of which is the fitting of earthing chains dangling on the floor to every insulated movable object in the operating theatre. Surgeons and anaesthetists become insulated movable objects when they wear rubber-soled boots and shoes ; all such footwear should have soles of conducting rubber, and boots of this sort are now obtainable. When the humidity of the theatre is kept as high as is compatible with working comfort the accumulation of static electricity is again less likely to occur.

Obvious sources of ignition abound in operating theatres. There are such old offenders as the sparking diathermy needle, and even the apparatus itself may be a danger, particularly when spark gaps are visible through a perforated metal case. These things must obviously be avoided in the presence of explosive vapours. Less well known is the risk associated with endoscope bulbs, which are so often overloaded because the surgeon wants more light. Ether vapour can be ignited by a temperature as low as 172° C., and these little bulbs may become very hot. Such a bulb may also break, with the momentary exposure of a white-hot filament. A simple device fitted to the voltage reduction apparatus will prevent their being damaged by overloading. Endoscopic instruments should also be inspected regularly to ensure that the lamp and the lamp-holder fit perfectly ; a defective contact at this point may be responsible for touching off an explosive anaesthetic mixture.[7]

Less obvious sources of ignition are numerous and cannot all be tabulated here. Surgeons often use appliances like electrical saws, drills, cauteries, and lights without considering whether these instruments are safe or otherwise from the point of view of setting off an explosion. No such device should be introduced into the theatre until it has been approved by a trained physicist or an engineer who could say whether or not there is danger of an explosion when the instrument is in use. In the same way an expert electrician should inspect regularly the switches and plugs in operating theatres. Loose connexions and deficient earth connexions can then be made good. It is perhaps hardly necessary to state here that open-flame sterilizers should never be used in theatres and anaesthetic rooms. Much has been written about all these and other precautions. Were they all adopted explosions would be very rare. But lack of awareness on the part of surgeons, anaesthetists, hospital administrators, and theatre sisters still continues. The surgeon who enters the anaesthetic room with a cigarette at his lips would not be welcomed in any factory where there was the same risk of explosion, and there are certainly some anaesthetists who would regard such conduct as "entirely inexcusable."[8] When an obvious source of ignition must be present expert anaesthetists can usually administer satisfactorily a non-inflammable anaesthetic. Thus in one explosion the diathermy needle was applied near the pleura in a patient who had recently had a pneumonectomy. The possibility of a bronchial fistula being present in such a case is well known, and the use of an explosive mixture like cyclopropane and oxygen in these circumstances is surprising. Chloroform, thiopentone, nitrous oxide, and curare, all non-inflammable, are commonly employed in thoracic centres all over the world, and it seems that in this instance the use of one of these might have prevented the explosion. In cases of this kind the diathermy point and the explosive vapour may be separated only by thin pleura and the surgeon's steady hand. It is by no means certain that the risk attached to non-explosive anaesthetics in such cases is greater than the risk of explosion.

Anaesthetic explosions continue to occur despite all the publicity that has been given to the subject in the medical literature and in the newspapers. It should perhaps be considered whether the precautions now generally accepted should not be made a legal responsibility of hospital authorities and of those working in operating theatres. Thus, deaths resulting from their non-observance might at least raise the possibility of salutary penalties. Interest in the prevention of explosions would then be given new impetus. When the Ministry of Health issued a memorandum[9] on this subject before the war it had to be addressed to medical officers of health and local authorities. The Ministry is now more intimately concerned than ever before with hospital activities, and might perhaps be stimulated to consider again the problem of anaesthetic explosions and the avoidable deaths which are caused thereby.

MALIGNANT HYPERTENSION

There is a growing conviction that malignant hypertension is a form of primary hypertension not preceded by structural renal damage but causing it. This is a result of careful clinical and histological studies of early cases, and recently Byrom and Dodson[1] have put forward experimental evidence which gives further support to the theory. In malignant hypertension acute focal necrotic lesions occur in the terminal arteries and arterioles of many organs, especially in the kidney, where the glomerular vessels are affected and tubular changes develop. Byrom and Dodson studied the onset of this acute necrotizing arteritis in the hypertensive animal and demonstrated the aetiological importance of increased intravascular tension: neither uraemia nor " toxic " absorption from damaged renal tissue was a necessary or constant precursor. Other writers consider that the characteristic arterial necrosis will not occur unless renal abnormalities are present—despite the finding of such necrosis in the early stages of the natural disease in patients with otherwise clinically and histologically healthy kidneys. Some even believe that the renal factors alone are concerned and that increased intravascular tension is not.

In their first series of experiments Byrom and Dodson used the Goldblatt technique to produce hypertension in eight young rats. The left kidney was rendered ischaemic by constriction of the left renal artery with a cotton thread ligature; the right kidney was undisturbed. The animals were killed after periods of 3, 7, 10, 14, and 17 weeks, during which time the systolic blood pressures were recorded. In every animal some atrophy of the ischaemic kidney was found, but no arterial lesion. There were necrotizing arterial lesions in the intact (right) kidney of six rats and in the heart, mesentery, brain, and spleen in a similar number. The finding by Schroeder and Neumann[2] and Patton and his colleagues[3] of necrotizing lesions in both kidneys can be explained as the result of variation in the ligation of the left renal artery. As Byrom and Dodson have shown, effective ligation acts as a barrier and protects the left kidney against the increased arterial tension. In an endeavour to find out how hypertension causes the arterial necrosis, they tested the hypothesis that a consequence of sudden overstretching of the vessel wall, as may occur when the intravascular tension is increased, is necrosis of the overstretched muscle and lysis; blood and plasma then seep into the affected zone and are transformed into a fibrinoid plug by the action of thromboplastin from the damaged muscle. Two millilitres of warm Ringer's solution was injected through a short bent needle (0.6 mm. bore) ten to fifteen times into the exposed left common carotid artery of 23 young adult rats; the injections were made towards the heart—the upper end of the artery being ligated. In 10 of the animals a loop of thread round the left renal artery was pulled taut during the injections, thus protecting the left kidney against the sudden increased tension. The transient rise in pressure which resulted probably amounted to between 80 and 90 mm. Hg. During the injections, which were made as forcibly as possible, the cortex of the kidney was seen to blanch momentarily and equally briskly to regain normal colour. After repeated injections, however, small irregular areas of residual pallor were noticed which returned to normal more slowly owing to transient spasm of the intralobular arteries. The animals recovered quickly and appeared well when killed three days later. Typical acute focal fibrinoid necrosis of the arteries of the cortex, including the afferent glomerular arterioles, was found, except in the kidneys protected by occlusion of the renal artery. A clear demonstration of the sequence of events leading to the appearance of the lesion was obtained—necrosis of a segment of muscle cells with autolysis, and with fibrinoid exudation into the adventitia and subintima with or without round cell and fibroblast infiltrations. Occasionally simple coagulative necrosis occurred. No hypertensive glomerular lesions were seen: the parenchymatous damage was limited to slight tubular degeneration—a clear indication of the brief duration of arterial contraction. This is a most important observation, since there is clinical belief that the acute symptoms in malignant hypertension, especially those associated with encephalopathy, may be caused by transient vasospasm.

Other experiments of Byrom and Dodson confirmed the findings of Leiter and Eichelberger[4] that bilateral renal-artery ligation causes earlier death (48 hours) than bilateral nephrectomy (76 hours), and that necrotizing arteritis (of pancreas and other viscera) may occur after ligation of both renal arteries or both ureters. This shows that the arterial and arteriolar lesion can arise in the absence of gross hypertension as a result of total renal insufficiency and total necrosis of the kidney. But this combination is never present in either experimental or human hypertension. Braun-Menéndez and his colleagues[5] suggest in an ingenious hypothesis that arterial necrosis may develop from a combination of two of the three factors—hypertension, uraemia, and the toxic absorption of dead renal tissue. But this does not explain the appearance of the arterial lesion in experiments where hypertension alone is present. While admitting that both uraemia and renal necrosis may play a secondary part in the " malignant " termination of chronic renal hypertension and may accelerate the late stages of true malignant hypertension, Byrom and Dodson interpret the results of their experiments as good evidence for the simple mechanical explanation of acute hypertensive vascular lesions. Their work supports the modern clinical concept of malignant hypertension and the assumption that the difference between benign and malignant hypertension is simply one of degree.

[1] *J. Path. Bact.*, 1948, **60**, 357.
[2] *J. exp. Med.*, 1942, **75**, 527.
[3] *Surg. Gynec. Obstet.*, 1943, **76**, 493.
[4] *J. clin. Invest.*, 1943, **22**, 11.
[5] *Renal Hypertension*, 1946, Springfield, p. 67.

ACUTE MASTOIDITIS IN CHILDREN

The sulphonamides and penicillin have changed beyond recognition the prognosis and treatment of acute infections of the middle ear cleft. Nevertheless each new chemotherapeutic substance has its drawbacks as well as its advantages. The drawbacks are not always apparent at first, as witness the masking of symptoms in acute mastoiditis and the increased risk of deafness from otitis media before this was recognized as a possible danger. It is now accepted

that these complications may largely be avoided by adequate early dosage and prompt drainage of pus. The latter may be unnecessary if chemotherapy is instituted early enough, but all authorities reiterate the need for drainage of pus. Elsewhere in this issue Mr. A. J. Moffett and Dr. G. A. Dalton report the results of treating acute mastoiditis in children with penicillin and sulphadiazine. They included in their series every case of undoubted acute mastoiditis seen in hospital over a period of three months. In addition to systemic disturbances, tenderness, swelling, oedema, and, in some cases, a fluctuating abscess over the mastoid process were present: the typical downward and forward displacement of the pinna was seen, to a greater or lesser extent, in all the cases. The children were treated in hospital with full doses of sulphonamides and penicillin in addition to local cleansing and penicillin aural drops thrice daily. Myringotomy was performed when necessary. Out of 17 children, whose ages ranged from 18 months to 12 years, treated in this way all except one were cured without surgical drainage of the mastoid. The otorrhoea ceased in an average of 5.6 days (limits, 3 to 11 days), the periostitis in an average of 8 days (limits, 5 to 11 days), and the drum recovered its normal appearance in 13 of the cases in an average of 10.7 days (limits, 8 to 19 days) from the beginning of treatment. In all cases the parents were written to several months after discharge of the child from hospital in order to find out if there had been any lighting-up of the infection, but none had occurred. The saving of suffering to these children and the reduction in time spent in hospital are obvious advantages, but the risks of treatment should be borne in mind. As the authors point out, a careful watch must be kept throughout for signs of intracranial suppuration, and before this method can be generally applied further trial should be made of it in hospital. There will always be need to take special care that the often ill-defined signs of continued suppuration or its intracranial spread do not go unnoticed. The possibility of using this method of treatment in adults may be considered, but it should be remembered that because of the greater vascularity of the temporal bone in a growing child the reaction to infection caused by organisms sensitive to chemotherapeutic substances will probably be more favourable in children than in adults.

AUREOMYCIN AND SYPHILIS

Supplies of aureomycin have until recently been scarce in this country, and little has therefore been published here on its use in the treatment of various diseases. The report by Dr. R. R. Willcox which appears on another page is probably the first to be published in England about the treatment of early syphilis with this drug. Willcox treated nine Bantu negroes suffering from primary or secondary syphilis with doses of aureomycin ranging from 0.75 to 1.5 g. over periods of 24 to 48 hours and noted that lesions, especially secondary ones, healed rapidly and that spirochaetes disappeared from surface lesions in 48 hours. Unfortunately follow-up examination was impossible, so that the effect on serum reactions could not be observed. One patient with secondary syphilis relapsed after 12 days. In the U.S.A. O'Leary and his colleagues[1] reported two cases of syphilis treated with aureomycin. The first, a case of secondary syphilis, was given 44.2 g. of the drug in 16 days; a mild Herxheimer reaction occurred after 24 hours. Spirochaetes disappeared in 60 hours, and a scrotal lesion healed in 26 days; but serum reactions, though weakening, were still positive after 41 days. Side-effects included nausea and anorexia. The second case treated by these authors was one of primary syphilis. This patient received 67.5 g. of aureomycin in 15 days; spirochaetes disappeared after 16 hours, and the primary sore healed in 15 days. Reactions were similar to those in the first case. In experiments on rabbits infected with *T. pallidum* 10 to 14 days previously Wiggall and his colleagues[2] found that intramuscular injections of aureomycin (50 mg. per kg. of body weight) caused lesions to heal and spirochaetes to disappear, though rather slowly. They also treated 15 patients suffering from primary or secondary syphilis with doses varying from 18 to 36 g. for two to six days. Again spirochaetes disappeared rather slowly, in some cases persisting for nearly 72 hours, but most lesions healed in six days. Side-effects were mild in men, but the drug was toxic for rabbits, possibly because it was given parenterally.

Aureomycin is best given orally in the form of 250-mg. capsules, and toxic effects in human beings appear to be slight, consisting only of nausea or vomiting, anorexia, and diarrhoea. The drug undoubtedly has an effect on syphilis, but its relatively slow action in killing spirochaetes suggests that it is not nearly so active a remedy as penicillin or arsenic. Its influence on serum reactions and its action in late, neuro-, and cardiovascular syphilis will not be known for some time. Meanwhile its use in the treatment of human syphilis must still be experimental, and no attempt to give it as a routine can be justified until more is known about dosage, period of administration, and possible toxic effects. In this connexion it is of interest to note that aureomycin is effective in all five venereal diseases—syphilis, gonorrhoea, chancroid, lymphogranuloma venereum, and granuloma inguinale.

[1] *Proc. Mayo Clin.*, 1948, **23**, 574.
[2] *Amer. J. Syph.*, 1949, **33**, 416.

Sir Herbert Lightfoot Eason, President of the General Medical Council, died on November 2. An obituary notice is published in this issue of the *Journal* at p. 1117. The next meeting of the Council over which Sir Herbert should have presided will open on Tuesday, November 22, at 2 p.m.

Dr. Donald Hunter will deliver the Ernestine Henry Lecture before the Royal College of Physicians of London (Pall Mall East, London, S.W.) on Tuesday, November 15, at 5 p.m. His subject is "Devices for the Protection of the Worker against Injury and Disease."

Dr. Norman B. Capon will deliver the Charles West Lecture before the Royal College of Physicians of London (Pall Mall East, London, S.W.) on Tuesday, November 22, at 5 p.m. His subject is "Development and Behaviour in Childhood."

REFRESHER COURSE FOR GENERAL PRACTITIONERS

MANAGEMENT OF THE PREMATURE BABY

BY

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According to official figures twice as many premature infants die each year in Great Britain as the total number of all persons killed on the roads. This indicates the seriousness of the problem. Half of these deaths occur in the first 24 hours of life and present a challenge to the obstetrician to prevent premature labour, but better management of the survivors would go far to reduce the loss of life. Although figures in connexion with prognosis are somewhat contradictory, the position can be summed up by saying that except for a slightly increased risk of cerebral trauma at birth the prematurely born infant has as good a chance as any other baby of growing into a normal and useful citizen.

Details of management to be discussed here are deliberately planned for what may have to be carried out in the patient's home. Some think this is a better place for premature babies than a hospital; others believe that the hospital can offer facilities and apparatus far superior to those available in a house. This should have been remedied to some extent since 1944,[1] when local health authorities were advised to keep certain appliances on hand for use in the home. A general practitioner called upon to look after a premature baby should certainly contact the medical officer of health for the district and see what apparatus he can get, and the services of a paediatrician should be available to lend expert help if required. More important than any apparatus and the aid of the expert, however, is good nursing. Some midwives are more skilled with small babies than others, and it may be possible to secure the services of one who has had special experience with premature babies—for example, at a maternity hospital with a special unit for their care.

General Management.

If premature labour has set in and a small baby is expected certain preliminary steps can be taken. It is wise to give the mother a dose of vitamin K. A cot or basket should be prepared with lined sides to prevent draughts. If time permits an oxygen cylinder should be obtained, with a close-fitting rubber mask or a nasal catheter for administration.

After the baby is born it should be wrapped in a warm towel and placed head downwards in the cot or basket, which should be suitably tilted. Gentle wiping out of the mouth and gentle aspiration with a rubber catheter will make sure that the air passages are clear. If available, oxygen may be administered for a short period to produce a good saturation of the arterial blood. If respiration is feeble carbon dioxide from a "sparklet" apparatus may be used, or some device improvised to secure a degree of rebreathing.

It must be remembered from the start that the greatest enemy is infection, and in the early days the nurse and doctor should wear an efficient mask and conduct all manipulations with an aseptic or antiseptic technique.

[1]Ministry of Health circular 20/44.

The room in which the baby is to be nursed must be kept warm—70 to 75° F. (21–24° C.) is ideal—in addition to the careful use of hot-water bottles in the cot or the employment of an electric blanket pad (which can be hired in some areas). The aim is to keep the baby's rectal temperature not below about 98° F. (36.7° C.), but this is sometimes not achieved for a few days. The temperature should certainly be taken and recorded four-hourly. The atmosphere must also be kept moist enough to minimize "evaporation" from the baby's skin and lungs. A wet towel hung over the back of the chair, with its end in a bowl of water and placed near a source of heat, will provide a reasonable degree of humidity. A bathroom, if available and able to be temporarily set apart for the baby, is often ideal because of its humidity.

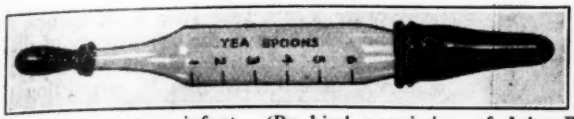
When the baby has recovered from the initial shock and is breathing well and a good colour the nurse may give it a gentle cleansing. This is most efficiently accomplished as a modified blanket bath with soap and water and a soft flannel, exposing only a small portion of the skin at a time. Drying should be carried out by gentle dabbing with a warm, soft towel, followed by a suitable baby powder (olive oil is messy, an inefficient cleaning agent, and not always sterile). The normal procedures for care of the eyes and cord should be carried out. Napkins can be applied, and the baby dressed in a gamgee jacket, bootees, gloves, and a bonnet, leaving only the face exposed. The bedclothes must not be too heavy. Before this dressing-up, however, the weight should be obtained if the baby's condition permits, for it is a most valuable base-line from which to judge subsequent progress. The infant should now be left in its cot and disturbed as little as possible, except to be moved from one side to the other from time to time to promote full expansion of the lungs. Oxygen may still be required at intervals if the colour does not remain good.

All these measures are necessary at first, but from the start the programme of management must be designed to speed up the process of maturation. That is to say, the baby must be brought daily nearer to the normal standards of care of the full-time infant. The external temperature of the room can be gradually lowered, some of the gamgee clothing cut away, and hot-water bottles or blanket pad left out of the cot for brief but increasing periods. It is difficult to lay down precise details to cover premature babies of all weights. Careful judgment is required, and the experience of the nurse is invaluable. I must stress the necessity for making progress and not keeping the baby too long before his own heat-regulating mechanism is called upon to take action.

Feeding

The same difficulty over precise details arises in connexion with feeding. Certain general principles will be discussed and, as far as possible, their application to a group of infants whose weights range from 2 lb. (1 kg.) or less up to the official standard of 5½ lb. (2.5 kg.).

Methods.—Most premature infants are too weak at first to suck at the breast, so that easier methods must be tried. In order of decreasing strain on the baby, after breast-feeding these are : sucking at an ordinary bottle ;



Feeder for premature infant. (By kind permission of John Bell and Croyden, Ltd.)

the use of a Breck feeder (a small bottle with a rubber nozzle at the end opposite the teat, like the old-fashioned fountain-pen filler, by which positive pressure can be exerted) ; a spoon or pipette to drop the feed into the mouth ; or tube-feeding by a catheter passed down the oesophagus through either the nose or mouth.

The first thing to establish after the baby is born is whether or not he can swallow. A little sterile water from a spoon can be tried. If there is no swallowing reflex, tube-feeding will clearly be necessary. If the baby can swallow, the next thing to ascertain is whether or not sucking can be carried out. If sucking proves possible a Breck feeder is the simplest apparatus: if not, a spoon or tube should be used according to the ability to swallow. The experienced nurse can soon determine a way of mixing the methods of feeding—one or two tube feeds and the rest by spoon, or starting the feed with a bottle and finishing with a spoon when the baby gets tired of sucking. Here, too, progress must be made and each day should see an attempt to take the baby a stage up the scale—tube, spoon, bottle—with the object of getting him to the breast.

Feeds.—Breast milk is the safest and most easily digested food. All premature babies should receive it at first even if it cannot always be continued. For the first 24 hours sterile water in small sips should be used, and during this period efforts to secure a supply of breast milk can be made. Only a few ounces a day will be required at first and should be obtainable without much difficulty from any nursing mother or from a maternity unit. It should be boiled and diluted to one-fourth strength for the very small babies and to one-half strength for the medium and larger-sized infants. Meanwhile the baby's own mother's supply of colostrum and milk should be obtained by manual expression.

By the end of a week or 10 days the breast milk can be given undiluted. If a supply proves impossible to obtain and no more can be got from the baby's own mother an alternative has reluctantly to be used. To avoid subsequent changes a choice should be made of a dried milk, either one of the so-called humanized varieties or a half-cream brand. These dried milks have the additional advantage of being obtainable at the local welfare centre. Either of these milks can be given from the second or third day of life and in a dilution which may vary from an eighth or quarter-strength, according to the baby's size, up to half-strength. One measure of milk powder to an ounce (30 ml.) of water may be assumed to be full strength. An attempt should be made to increase the strength daily, aiming eventually—in the second or third week—at full strength for the humanized brand and three measures to 4 oz. for the half-cream variety.

For simplicity, the addition of substances to the milk is not recommended. Some authorities prescribe additions of soluble casein and others prefer to use evaporated milk, sweetened or unsweetened, but the programme outlined above works well and, as has been said, minimizes changes when the infant is older.

Times of Feeding.—Quantities and intervals of feeding are related. It must be remembered that small babies require relatively more food than full-term infants. If the basis for a 7-lb. (3 kg.) baby is 2½ oz. of breast milk per pound (150 ml. per kg.) body weight per day, then the smaller infant may require as much as 3, 3½, or even 4 oz. per pound before a steady weight gain is achieved. But, as with normal babies, such a full feed programme should not be attained too quickly. If the largest feed, in volume, is achieved by the seventh to tenth day, this is usually safe. If dried cow's milk is being used progress should be slower and for the smallest babies the full size (and strength) not reached until a little later. Provided that no oedema develops the weight curve is a most valuable guide and the experienced nurse can increase the strength and size of the feeds to keep pace with the strength and requirements of the infant. With the smallest infants this will usually mean beginning with feeds of 30 to 60 minims (1.8–3.5 ml.) every hour by day and two-hourly by night. Gradually this is increased to ½ oz. two-hourly by day and three-hourly by night, until 1 oz. is taken every three hours round the clock. If the mother's milk supply has been established the infant may be tried at the breast at least once a day if over 4 lb. (2 kg.) in weight at birth, or when this weight is achieved. Even smaller babies, especially if twins (and therefore relatively more mature than the weight indicates), can suck quite strongly under this weight.

Whatever the feed a start should be made with vitamin supplements when the infant is a month old. Vitamin C as ascorbic acid in 25-mg. daily dosage can safely be given even before this. Vitamins A and D are best given as a vitamin concentrate to allow 1,000 to 2,000 units of vitamin D daily, and such dosage—twice the requirements for a full-term baby—should be continued throughout the first year. Iron is also best administered early, say at two to three months, and given in Mackay's mixture in a dosage of 1 gr. (65 mg.) three times a day.

Two other medicaments are also worthy of mention. Some paediatricians give all babies vitamin K after birth to prevent hypoprothrombinaemia, and, as already suggested above, if premature labour is anticipated this vitamin can be given to the mother also. Thyroid extract has a useful part to play. If the baby is particularly sluggish and difficult to rouse and has a subnormal temperature, the metabolism can certainly be stimulated by giving the equivalent of 1/10 gr. of thyroid extract per pound (14.4 mg. per kg.) body weight daily. This should generally be administered for a few days only, after which the increased food intake will itself stimulate metabolism.

Thyroid extract is the only safe stimulant for the premature baby. Brandy appears to be used fairly frequently, but it is irritating to the stomach and intestines and if given in stimulating dosage may produce a dangerous gastro-enteritis. If the baby is kept adequately warm and gently handled brandy should be unnecessary. If anybody needs it, perhaps the doctor and nurse should be considered.

A circular to hospital boards and committees from the Ministry of Health asks hospital management committees to ensure that medical superintendents of mental hospitals and mental deficiency institutions see a copy of all documents on the work of the hospitals and institutions issued to the committee by the Ministry or the Board of Control. It also warns hospital management committees that they should not alter the authorized accommodation in mental hospitals and mental deficiency institutions without the approval of the Ministry of Health.

[The Times

ROYAL COLLEGE OF OBSTETRICIANS AND GYNAECOLOGISTS

QUEEN ADMITTED HONORARY FELLOW

The Queen was admitted an Honorary Fellow of the Royal College of Obstetricians and Gynaecologists on November 3 by Sir William Gilliatt.

Sir William Fletcher Shaw, past President and first Honorary Secretary to the College, and Professor R. W. Johnstone, Senior Fellow in Scotland, presented the Queen for admission.

Sir William Fletcher Shaw said that, as the College had developed and matured and its value to the welfare of the women of the Empire had been more fully established, her Majesty had very graciously become the first Patron of the College. "That your Majesty accepted this office gave great joy to the College and added immensely to its prestige in all the countries of the Empire." More recently her Majesty had shown her great personal interest in the work of the College by the princely gift she had made to its Appeal Fund. "Now by becoming an Honorary Fellow of the College and thus identifying yourself with it in an even more personal way you confer the greatest possible honour on the College."

Professor Johnstone said: "When you, Sir, were kind enough to award me the great and happy privilege of acting as one of her Majesty's sponsors on this occasion, you remarked to me that her Majesty was a Scotswoman and was very proud of it, and that it would be becoming that one of her sponsors should be a Scottish Fellow of the College. In regard to that I would only say that her Majesty's very natural and proper pride of race meets an overflowing complement in the deep and affectionate pride which every Scotsman and Scotswoman the world over takes in her Majesty's most gracious self." All at the College were well aware that the College was receiving a much greater honour than it was giving.

After expressing her gratitude to the College, the Queen said that she shared with all her heart their belief that it was on the happiness of home and family life that the true worth and strength of a nation depended, but what had been so well described as "the miracle of life and the source of all tenderness and love in humanity" demanded more than ever the best technical attention. "It demands a spiritual approach as well, and it is good to know that in your profession its members bring to their work a warm human understanding and a deep sense of moral values."

Handing the Queen her scroll as a Fellow, Sir William Gilliatt said: "May it please your Majesty that I confirm your two sponsors in that the addition of your name will add distinction and lustre to the roll of Honorary Fellows.

"We are aware that your Majesty has never had, and cannot have, the opportunity of directly advancing the scientific side of our subject. I maintain, nevertheless, that by permitting us to elect you an Honorary Fellow and by your very generous gift to our Appeal Fund you are indirectly advancing in no uncertain manner the important work in which we are engaged. Your gracious acceptance of our Fellowship encourages us to feel that your Majesty is showing an even closer interest in our activities than was possible during the years you have been our Patron.

"We could not have asked you, Madam, to join us did we not consider that there are few higher aims than to contribute to the prevention of illness and the maintenance of health and well-being of mothers and their infants. Your Majesty's sponsors have spoken of the standards of family life in which you have set the world such a wonderful example. In our report on 'A National Maternity Service' published in May, 1944, we use the words, 'We are all conscious that the infusion of higher moral and spiritual values into the families of our country has a profound bearing on physical and mental health.' We on our part are encouraged by your presence here to-day and are determined to show that your trust in our future achievements has not been misplaced.

"May I, on behalf of Council here present and every Fellow and Member, welcome your Majesty as an Honorary Fellow and thank you for your gracious acceptance."

New President Installed

Installing Professor Hilda Lloyd as President, Sir William Gilliatt said that it gave him great pleasure to be the first to congratulate her on assuming the high office to which she had been elected. Her election was an outstanding occasion in that she was the first woman to be elected as President to one of the Royal Medical Colleges. He offered her, on behalf of his colleagues on the Council and for himself, their best wishes for very successful years of office. He wished to record his deep appreciation of her generous action in proposing to and convincing the Council that his term of office should be prolonged so that he might have the privilege of admitting her Majesty to the Honorary Fellowship of the College.

Professor Hilda Lloyd was then vested with the president's gown and repeated the president's oath.

Mr. V. B. Green-Armytage and Mr. J. Eric Stacey were installed as Vice-Presidents in place of Sir William Fletcher Shaw and Mr. James Wyatt, whose term of office had expired.

The Handicapped Pupils and School Health Service Amendment Regulations, No. 2, 1949, make certain interim amendments to the main regulations recommended by the Education Subcommittee of the Local Government Man-power Committee. It is now unnecessary for the local authority to obtain approval of the arrangements made or proposed for carrying on the school health service; the Minister will rely on inspection and on the specified annual returns. The approval of medical officers is limited to those employed in connexion with the authorities' arrangements for ascertaining whether pupils require special educational treatment because of educational subnormality. A number of forms have also been withdrawn because the National Health Service has made them obsolete. Under the Reports to Local Authorities (Records) Amending Regulations, 1949, one of the statutory forms on the reporting of children to the local health authority has been amended to facilitate co-operation between the educational psychologist and the medical officer in order to obviate duplication of work.

GROWTH AND REPRODUCTION OF BACTERIA

Lectures by Sir Paul Fildes

A course of special University of London lectures on general bacteriology was given at University College in October by Sir PAUL FILDES. His account of the growth and reproduction of bacteria covered a wide range, and it is possible to record only certain points of especial interest.

Sir Paul Fildes said that he intended to try to analyse some of the means by which bacteria were maintained as part of the population of the planet. This involved a description of a number of biochemical processes which were not peculiar to bacteria but were common in some degree to all living things. It was hardly possible to lecture on the biological processes of bacteria without making it obvious that bacteria were being used only as illustrations of cellular physiology in general. He might have selected other organisms, such as protozoa or plants or animals, but bacteria were chosen because they were easy to handle and yielded results within a matter of hours. It was for this reason that many of the findings of cellular physiology had been based upon the study of bacteria.

The physiology of bacteria derived its special importance from the likelihood that the control of bacterial activity in health or disease would not be achieved without greater knowledge than was available at present. Bacterial activity became apparent only as a sequel to growth and reproduction. This occurred automatically when conditions were favourable. Growth and reproduction were therefore the prime interests of cellular physiology, because without them the external effects of bacteria for either good or ill would be unimportant.

Organized Enzyme System

It was convenient, Sir Paul continued, to look upon a bacterium not as a mere "bagful of enzymes" but as an organized enzyme system. The organization was such that when the whole system was steeped in the proper substrates the "machine" started to work and continued automatically until the substrates failed or the environment became adverse. The co-ordinated working of the system resulted in an increase in the contents of the bacterial cell until there was a division of the cell into two. When the process was working in full co-ordination—which was not always the case—the time taken by one cell to become two might be some 30 minutes, so that in 24 hours there might be 48 generations and the one bacterium would become 17,000,000.

If nitrogen were taken as the key substance of the protein molecule, it was known from the work of Behring that some bacteria grew in gaseous nitrogen while some grew in such forms of nitrogen as ammonia. As the yield on analysis showed a vast increase in protein, it followed that protein was synthesized from ammonia by the organism. The first thing that happened was that the ammonia diffused into the cell, then it was fixed by one of the receptors of an enzyme and combined with another nutrient fixed by another receptor. In general the synthesis of protein might be looked upon as a chain of reactions carried out by a series of enzymes, each of which had two receptors and two substrates. Eventually by the serial combination of two similar substances the whole protein molecule was constructed.

Genetic Mutations

In order to bring bacteriology into line with general biological teaching it was necessary to make an assumption which could not yet be proved. It was held that the controlling or operating force behind the enzyme was a gene, and that genetic mutations led necessarily to changes in or the disappearance of enzymes in bacteria. These phenomena might have a marked effect on the survival of bacteria in particular environments.

A great deal of work had been carried out in elucidating the manner in which proteins were synthesized, and it was possible to make some tentative guesses at the steps of the synthesis. He described the role of tryptophan as a growth factor, also of indole, which could take the place of tryptophan. It was a question of guessing what two substances might be concerned in the synthesis of indole, for example, and testing whether they would have any effect in the absence of indole. Until an organism could be found which would give a positive growth response to a hypothetical growth factor, guessing could go no further.

The action of a synthetic enzyme was the reverse of that of a degraded enzyme. Energy was absorbed by the former, liberated by the latter. A synthetic enzyme combined two substrates into a larger, while the other type of enzyme split a larger into two smaller substrates. One and the same enzyme was capable of operating in both ways.

In his final lecture Sir Paul Fildes said that it was possible to summarize the two views—the former and the present view—as follows. When the structure of the bacterium altered, whether by the gain or the loss of an enzyme, it was considered to be due to some stimulus deriving from the environment. Broadly, it was said that, if a bacterium found itself in an environment rich in some particular substance which normally it had to synthesize, it did not synthesize and the enzyme associated with the synthesis faded away. The geneticists explained the phenomenon differently. They developed the generalization that behind every enzyme there was a gene which controlled its action. In the course of the reshuffling of genes in the process of cell division accidents might happen, in the same way that accidents might happen in any process which was often repeated. That was merely a matter of statistics. If an accident happened to a gene which affected its normal function the enzyme would be affected. In a normal case with the enzyme present it might disappear, or, if the enzyme was absent from the particular organism being studied, mutation occurred which had nothing to do with environment. The main conflict was whether mutation was merely a matter of chance or was induced by outside causes.

When inoculations in agar without tryptophan were made, a normal typhoid bacillus, for example, did not grow at all at first, but, as the size of the inoculum on the plate continuously increased, the time came when one colony appeared, and more still appeared with further increase of size. If one of these colonies were picked off and tested on the liquid medium it would grow normally and maintain the difference indefinitely. In every culture of typhoid bacilli there were a few abnormal units growing in a different manner. These abnormals were not necessarily present in the original inoculum; they appeared as the culture grew.

Environmental and Accidental Factors

The fact that these mutants made their appearance at widely different points in the growth of the culture, and sometimes not at all, indicated an erratic spontaneous origin rather than the constant influence of the environmental factor. Formerly it was assumed that the environment was concerned mainly with these mutations, but it now seemed reasonably certain that the general idea accepted in genetics held good, that when an operation like cell division was repeated often enough the liability to "accident" increased and there were more likely to be missing or altered genes.

Another series of observations also suggested a genetic origin. It was now well established that exposure of bacteria to constant doses of *x* rays or ultra-violet light caused an increase in the mutation rate of the survivors. This meant that the survivors would produce mutations perhaps at every hundred divisions instead of at every million. The mutants did not occur in the absence of some growth factor. The *x*-ray effect was thought to be due to a filtered chemical action on a single molecule—a one-cell action, the one cell being enough to produce mutation. One of the chief reasons for holding this "one-cell" view was that the number of mutations produced was proportional to the dose of *x* rays.

In sum, therefore, there were reasonable grounds for suggesting that mutations in bacteria and genetic mutations in higher

forms of life had many points in common. They appeared at random and spontaneously, whereas they did not occur following specific changes in the environment. The new character was as stable as the original. In addition to this kind of evidence favouring the existence of genes in bacteria, a more simple argument could be brought forward. No one could imagine that the bacterium was a bag with all its molecules in solution; if this were the case there would be little likelihood of the molecule finding itself on the right side of the curtain when division took place. There must be some mechanism in bacteria as in other cells by which characters were segregated in special divisions.

Sexual Process in Bacteria

It was often supposed that bacteria were non-sexual. Here he recalled an experiment in which a culture of *Bact. coli*, which grew quite well on ammonia because it was able to synthesize all its necessary requirements, was exposed to *x* rays to produce mutants. From the resulting mixture two strains were selected neither of which would grow on ammonia for the reason that one of them was unable to synthesize, say, substances A and B, whereas the other was unable to synthesize substances C and D. Starting with two strains, one of which had lost the genes controlling the A B synthesis and the other the genes controlling the C D, a strain was produced with all the genes and no growth disability. This could have occurred only by the fusion of one cell containing the A B substance with another containing the C D, producing a mutation which contained all four.

The fusion of two cells with reshuffling of the genes was the basis of sexual processes by which hereditary characters were handed on. In animals evolution had produced two entirely separate types of cell, each with complementary genetic disabilities, so that they could not reproduce without fusion. On the other hand, in bacteria this specialization had not taken place. Usually each cell had an adequate genetic establishment, and was developed to allow reproduction by fission in the environment in which it happened to live. At, say, every 20 generations, however, mutations would appear and cells would be produced with different hereditary characters. If two such mutations happened to take place at the same time, and if, more rarely, they happened to fuse and produce a strain of rearranged genetic pattern, and if, finally, this new pattern was more satisfactory for survival in the environment, this would be an established mutation due to sexual process. Necessarily this must be rare. In animals the opportunity existed permanently, and was taken advantage of at short intervals, and for this reason animals had evolved by natural selection more than had bacteria. In general bacteria did not differ, so far as could be judged, from their earliest progenitors.

The argument for including bacteria among the sexed population of the planet was cogent. There could be no doubt that bacterial culture was liable to occasional change in a random manner without any known determining change in the environment. The lecturer pointed out the importance of this in practical work. A bacterial culture could always be looked upon as a population of similar type yet continuously subject to innumerable chance mutations of any of its characters, and any one of those mutations might be selected by the medium which was being used.

The idea of permanent alteration of character resulting from genetic mutation could not yet be correlated with many known facts about bacterial enzymes. In particular, genetic ideas could not be correlated satisfactorily with what was known about the enzymes described as adaptive. Some enzymes had been known to increase in activity or to appear from apparently nothing when brought into contact with a specific substrate, without any cell division at all. This was clearly a phenomenon different from genetic mutation, but it was not a permanent change. It faded away when the substrate was removed, and it was evidently due to environment and not to chance. The vast field for speculation along these lines, as Sir Paul Fildes pointed out in closing his course of lectures, indicated how many matters in the field of bacteriology awaited further research.

Reports of Societies

WHITHER ANTENATAL CARE?

A discussion on this subject took place in the Section of Obstetrics and Gynaecology of the Royal Society of Medicine on October 21, with Dr. LESLIE WILLIAMS presiding. Reference was first made to the election of Professor Hilda Lloyd to the presidency of the Royal College of Obstetricians and Gynaecologists. This was the first time a woman had been elected to the office, and she had gone straight to the position from the presidency of the Section.

Mr. ALECK BOURNE said that to debate the future it was necessary to review the past. The story of antenatal care was a chequered one. Until the middle of the last century no real interest was taken in the examination of the pregnant woman from the point of view of her health. In Great Britain the first clinic for providing supervision and advice for expectant mothers was that established by J. W. Ballantyne in Edinburgh in 1902, but Ballantyne was occupied with the prevention of stillbirths, monsters, and deformities. He was not an obstetrician, and his interest in the health of the pregnant woman was only in so far as it might affect her baby.

The progress of antenatal care was greatly stimulated by the first world war. In 1918 antenatal clinics began to be established, and now there were over 1,600 in the country. They were staffed mostly by antenatal officers who did not conduct the labour. Thus there had been four phases in the progress of antenatal care. The first dated from ancient times. Aristotle's instructions to midwives included such fantastic remedies as the following: "Take a swallow's nest and dissolve in water, strain, and drink it warm, and it gives delivery with great speed and much ease," but there was no mention of the examination of pregnant women. The second was in the latter part of the nineteenth century, when the care of pregnant women from the point of view of eugenics was suggested. The third, associated with Ballantyne, was concerned with the quality of the babies born. The fourth, within his own memory, starting about 1909, was concerned with the discovery of conditions which were likely to make labour difficult. In those days it was uncommon to examine a woman more than twice, towards the end of pregnancy, and no interest was taken in her general health, her personality, or her social surroundings.

This led to an immense amount of interference. Obstetricians became obsessed with the possibility of a difficult labour. If the head was high it was assumed that it would not fit into the brim. G. E. Herman, at the London Hospital, published a book, which was still of great interest and value, entitled *Difficult Labour*, and in the fifth edition (1910) he gave a number of dates from the thirty-sixth week onwards at which induction of labour should be undertaken. Watts Eden published a paper in which he pointed out that the child born from the thirty-sixth to the thirty-ninth week was just as well able to survive as the child born at the fortieth week, and this gave a considerable stimulus to the induction of labour. In Queen Charlotte's Hospital labour was induced in the following percentages of all admissions:

1910	.. 4 per cent.	1920	.. 5.3 per cent.
1912	.. 2.5 „ „	1921	.. 7.4 „ „
1913	.. 5 „ „	1922	.. 6.6 „ „

Between 1928 and 1934 the maternal mortality rate was steadily rising, and in the latter year reached 4.41 per 1,000 live births, the highest figure since the 'nineties of the last century, and this after 15 years of increasing antenatal care and improved teaching of students and midwives. It was not surprising that critics began to point out that antenatal care had failed in its object of reducing the death rate in child-bearing. It was gradually realized that external measurements were useless, but unfortunately the mechanists had another lease of life as a result of *x*-ray pelvimetry.

Throughout the whole range of medicine, Mr. Bourne continued, there was a tendency to confine the supposed normal within too narrow limits, and this was more marked in obstetrical

work than in any other branch. One example was the use of pelvic measurements in the prognosis of labour. Even with reduced measurements there might be a difficult labour only if the uterine contractions were feeble; there were few complications which did not lose something of their potential danger if the uterus reacted strongly. If function was so important, was it possible to predict it during pregnancy, and, if it was, could anything be done antenatally to ensure the maximum possible function? In many cases he thought this could be done. He believed that the function of the uterus depended primarily on the emotional attitude of the woman in labour, chiefly her degree of anxiety. It depended also upon her clinical or biological type. The vast majority of women could not be divided into one or other of the extreme types—the functionally good reproductive type and the difficult type—but it was possible with insight and experience to say on which side of the middle line they were.

The function of the uterus could be predicted by watching the emotional attitude of the woman to her forthcoming labour. Many began their pregnancy in ignorance and went through it in uncertainty and fear, and their fear was such that it was practically certain they would have inertia, maximum pain, and an assisted delivery. Antenatal visits afforded an opportunity for watching the state of mind and understanding the emotional state. Sometimes the condition was masked by a spurious courage, but it was possible to break that down and see what lay underneath. Nothing could be done to alter things physically, but a great deal could be done to exorcise fear and liberate uterine function from the inhibitions which fear imposed upon it.

On the question of diet, Mr. Bourne suggested that there was no evidence as yet that malnutrition had an effect upon uterine contraction, though it certainly had in precipitating early labour. As for exercise, its chief value was in making the woman feel that she was contributing something to the preparation for her labour.

Elimination of Fear

Professor W. C. W. NIXON said that he was a student of Mr. Bourne's a quarter of a century ago, and recalled his teaching on the necessity for a shift of emphasis from structure to function. Unfortunately, the mechanistic fog still existed, but there was an awakening to the need for taking the woman as a whole, considering both *psyche* and *soma*. The normal pregnancy had not the drama of the abnormal, and the assistant and student gaining experience voted a week without an incident as rather dull.

In his opinion there should be a food advice bureau attached to every antenatal clinic. Nutritional anaemia was still prevalent, and the only way to detect it was by an estimation of the haemoglobin. Every pregnant woman should have her haemoglobin estimated on her first visit and again at the thirty-sixth week. Investigations had shown that the idea that excessive weight was an indication of toxaemia was a fallacy. During the last few years at the antenatal clinic at University College Hospital radiography of the chest had been done as a routine, and the results had justified the procedure.

At University College Hospital it was felt that by the introduction of training classes much had been done to eliminate fear. In these classes the changes in pregnancy and the birth process were explained, and the mother was given confidence and enabled to recognize the various events of labour as signposts rather than as hidden enemies. It was Dr. Grantly Dick Read who had first pointed out the importance of the tensions set up by fear and how these could be relieved by reassurance and relaxation. Everyone recognized the need for mothers to be physically fit so that they could pass through labour relatively unharmed. It was important also to prevent mental trauma. If this were neglected the mother was denied an enriching and satisfying experience.

Professor Nixon then showed a film, taken in his out-patient department, illustrating the type of physical exercises taught to expectant mothers, and also the work of the food advice bureau. The film had been specially prepared for the meeting by Mr. P. L. C. Diggory, film secretary of the British Medical Students Association.

Value of Exercises

Mr. A. P. WRIGLEY said that at long last the general health of the patient was receiving attention. Energetic measures had been adopted to prepare the mother physically and mentally for labour, the physical side being dealt with by remedial exercises, proper food, and teaching of correct stance and relaxation. The first results were beginning to come in and were not as encouraging as might have been hoped. The statistical difference between patients who had had exercises and patients who had not did not appear to be marked. In a further attempt to help patients at St. Thomas's physiotherapy had been combined with a mild form of psychotherapy, but an analysis of 106 women who had elected to join these classes, when compared with a number of controls, seemed to show no advantage. No one regretted this more than he did, although the sister told him that these patients were in a much happier frame of mind when they arrived for labour. He had no doubt that this work was in its infancy, and would show better results as experience accumulated.

He thought there should be a much more common-sense outlook about the expected date of delivery, and less, not more, attention should be given to the pre-natal preparation of the patient for breast-feeding. If on a casual observation the breasts appeared normal and the woman accepted breast-feeding as the natural thing, he regarded any treatment of the skin or massage of the breast as unnecessary and undesirable. The position with regard to *x*-ray examination had changed during the last ten years as a result of intensive and painstaking observations. The technique of radiology was now such that risks of misinterpretation had decreased. The day was not far distant when an *x*-ray picture would be made of the pelvis of every woman embarking on her first pregnancy, and this would be done by someone trained for that work.

He pleaded also for a less rigid adherence to routine—routine in attendance or in the procedure at the clinics. Patients differed from one another right down to the last weeks of pregnancy. The lives of patients had become so regimented that if the burden could be eased by less adherence to schedule and more use of common sense it was the doctor's duty so to ease it. Finally he spoke of the liaison between outside antenatal clinics and those who had the care of labour. Instead of drawing them nearer, the effect of recent legislation was to drive them further apart, and as long as the present system continued the care of the woman remained divided. Altogether, while the results of the last 15 years had been good, he was certain they could do better.

In some brief general discussion Dr. ARNOLD WALKER said that he thought there should be a little less criticism of what the local authorities had done in the antenatal clinics, for, after all, this was the only way in which many women could get antenatal care, and the closing of many such clinics created a gap which it would be difficult to fill. Another speaker pointed out that, whereas at the hospital clinics there was no guarantee that the patient would see the same doctor again, at the local authority clinics the people worked as a team and also had a broader outlook on the background of social medicine than their colleagues in hospital. Dr. HAROLD WALLER said that mastitis was almost a preventable disease, but in some clinics it was as high as 10% among lactating women.

Mr. ALECK BOURNE, replying, said that he wondered whether it was possible to relate the benefit of antenatal physical exercises to a statistical presentation of abnormalities at labour. One value of the exercises was to give a woman a positive attitude towards her own effort.

HYPOPITUITARISM

At a meeting of the Liverpool Medical Institution on October 13, with the president, Professor Charles Wells, in the chair, Dr. V. K. SUMMERS read a paper on the syndrome of hypopituitarism.

Dr. Summers said that the commonest cause of severe chronic hypopituitarism was necrosis of the anterior lobe at the time of delivery. These patients were in a state of severe circulatory collapse, and this collapse was produced usually by a

combination of shock and haemorrhage; the obstetric conditions likely to be associated with pituitary necrosis were postpartum haemorrhage and retained placenta. The symptoms and signs were due to a failure of secretion of pituitary hormone. The first sign was failure of lactation, and in these cases no milk could be expressed.

Nutrition was unimpaired in these women, and the majority were, if anything, slightly above average weight for their height and age. The term "pituitary cachexia" used for this syndrome should be abandoned, because it was misleading and the source of many errors in diagnosis. Failure of menstruation with severe atrophy of the whole of the genital tract was an invariable sign.

Signs of adrenal insufficiency were constant; electrolyte changes which resembled those of Addison's disease, but of less severe degree, occurred. In contrast to the heavy deposition of pigment in Addison's disease, a depigmentation of exposed areas of the body and of normally pigmented areas—for example, the areolae—was seen in hypopituitarism. Loss of pubic and axillary hair was always noted within two years of the onset of the disease. The excretion of ketosteroids was reduced to very low levels. Loss of thyreotropic hormone secretion produced symptoms like those of hypothyroidism: a dry skin, low basal metabolic rate, elevated blood cholesterol, sensitivity to cold, and cardiac changes resembling those of myxoedema. Cardiac enlargement did not appear, and the scalp hair remained normal in amount and texture.

Professor H. L. SHEEHAN said that the anterior lobe of the pituitary could be destroyed by a variety of pathological lesions. The most important of these was post-partum necrosis, which accounted for about three-quarters of the cases of severe destruction of the gland. The loss of anterior lobe hormones produced a great atrophy of the adrenal cortex, the thyroid, and the gonads, and the loss of hormones from the gonads led to atrophy of the genital tract.

Dr. C. O. STALLYBRASS referred to the work of Dougherty and White on the activity of the adrenotropic hormone in stimulating the mechanism of antibody production and, more especially, lymphocyte and plasma cell destruction with consequent liberation of antibody. If the adrenal cortex was gravely affected, as in the Friedrichsen–Waterhouse syndrome in meningococcosis, then a twofold lethal effect would be produced. He also referred to veterinary interest, described by Kay, in the action of the lactogenic hormone in assisting cows to "let down" their milk, and the experiments that had shown that while the stimulus might be neural the mechanism was hormonal.

Nova et Vetera

DISCOVERERS FOR MEDICINE

Discoverers for Medicine. By William H. Woglom, M.D. (Pp. 229; illustrated. 18s.) London: Geoffrey Cumberlege. 1949.

The purpose of this unusual book is given in the author's quotation from Oliver Wendell Holmes—"Medicine learned from a Jesuit how to cure ague, from a friar how to cut for stone, from a soldier how to treat gout, from a sailor how to keep off scurvy, from a postmaster how to sound the Eustachian tube, from a dairymaid how to prevent smallpox, and from an old market woman how to catch the itch insect." The author describes these and many other debts which our profession and its patients owe to the traditions and common sense of the unlearned and to the patient researches of scientists whose work and results seemed to have no bearing on medicine. With an easy style and infinite patience Dr. Woglom traces to these sources the benefits of vaccination, of digitalis, of quinine, of spectacles, and of many other aids and remedies which we now take for granted without a thought for their obscure beginnings. The book is handsomely produced, with many good illustrations and a copious bibliography and index. In illuminating the background of his discoverers the author does so with vivid sketches of the personalities of his heroes. ALFRED COX.

Correspondence

British Branch of Ligue Internationale Contre le Rhumatisme

SIR,—The recent International Congress on Rheumatic Diseases in New York at which Hench and Kendall presented their paper on "cortisone" was sponsored by the Ligue Internationale Contre le Rhumatisme, and there can be no doubt in the minds of those who heard this paper and availed themselves of the varied opportunities at this congress of the immense value of such meetings.

We feel that the British Branch of the Ligue is not sufficiently widely known and that membership offers not only individual advantages but an opportunity to foster friendly international relations at these various gatherings held in different countries. The next congress of the Ligue Européenne is in Barcelona in 1951, and the whole Ligue Internationale will meet in Zurich in 1953. Judging by the high standard of papers in New York this year, these two promise to be important meetings.

Individual membership of the British Branch entitles attendance at these congresses and also at certain clinical meetings of the Heberden Society. The British Branch is arranging a quarterly publication of a pamphlet giving news of forthcoming meetings of interest, of postgraduate courses, and a list of clinics where postgraduate members will be welcome.

The annual subscription is one guinea and should be sent to the treasurer, Dr. G. D. Kersley, 6, The Circus, Bath.—We are, etc.,

London, W.C.1.

W. S. C. COPEMAN,
President, Ligue Européenne.
OSWALD SAVAGE,
Secretary, British Branch of the Ligue Internationale Contre le Rhumatisme.

Progress in Obstetrics

SIR,—Since you did me the unexpected honour of using some of the subject matter of my Joseph Price Oration in America and my presidential address to the Edinburgh Obstetrical Society as a sort of text for your leading article on "Progress in Obstetrics" (October 8, p. 799), I feel it is incumbent on me to take some part in the discussion that has followed.

I am almost in entire agreement with the views advanced by Professor Andrew M. Claye (October 22, p. 927), which vary little from the suggestions I advocated in my Edinburgh paper. There is little doubt that the teaching of obstetrics has advanced greatly during the last twenty years, and, as I pointed out, I considered that this was one of the several reasons which had helped to lower the maternal mortality and morbidity. But even now, in my opinion, the newly qualified graduate is seldom capable of undertaking midwifery in a general practice until he has acquired more experience. This is not due to the teachers of obstetrics or to the students themselves, but to the paucity of cases available to give each student a thorough grounding in such everyday procedures as forceps delivery, the suture of the torn perineum, episiotomy, etc., not to mention the more difficult manœuvres, such as the turning of the occipito-posterior, external version for a breech presentation in a primipara, etc. It would therefore seem to be necessary to conserve cases for those who desire to practise midwifery in their general practice, and not to provide a number of midwifery cases to those who are later not going to be general practitioners but surgeons, pathologists, bacteriologists, psychiatrists, etc.

I suggested that if a year was to be spent following the final examination for clinical work before a degree was conferred those hoping to practise midwifery in the future would spend at least three months resident in a maternity hospital, where they would get an opportunity of carrying out the procedures I have already mentioned and at the same time obtain further clinical experience in normal and abnormal midwifery. In order to make cases available I suggested that it might be quite sufficient for the ordinary undergraduate to attend five

normal cases during his obstetric course and that residential stay in a hostel attached to the maternity hospital to study abnormal midwifery would be unnecessary. This would give the G.P. obstetrician-to-be much more material wherewith to acquire a better knowledge of the subject, and hostel accommodation in or near a maternity hospital would be available. Accommodation and the required tuition could be supplied not only in the present teaching hospitals but in non-teaching hospitals all over the country as well, on whose staff there was an obstetrician qualified to teach such students. A practitioner who had not previously taken this extra instruction and who wished later to do midwifery could take such a course at any time. In order to know which doctors were suitably qualified a diploma, such as D.Obst., might be given at the end of each course to all those who had acquitted themselves to the satisfaction of the consultant in charge, or after passing a small clinical examination conducted by an outside panel of examiners.

I quite agree with Dr. Walter Radcliffe (October 22, p. 927) that permission should be granted to general-practitioner obstetricians to attend patients in a number of maternity hospitals in the country provided they obey the rules of the hospital concerned and their work is supervised by the consultant in charge in the same manner as all the rest of the cases in the hospital are. This plan is carried out in a large number of the maternity hospitals in the U.S.A. with most satisfactory results, the practitioners being on what is called the "courtesy staff," and I may state that such facilities are already accorded to practitioners by at least one regional board in this country.—I am, etc.,

Edinburgh. W. F. THEODORE HAULTAIN.

Natural History of Asthma

SIR,—It is a long time since I practised medicine and I should not dream of criticizing the article by Drs. Elsie O. Williams and G. E. O. Williams (October 22, p. 897). At the same time, I cannot help feeling that it falls short, in some ways, of its majestic title.

My late friend, James Adam, of Glasgow, was interested in the subject precisely from the point of view of "natural history" and I doubt if he would have approved an essay which took so little account of habit and environment. In his search for clinical findings, too, he was more fortunate than your contributors. For example, he found eosinophilia almost universal and a consistent lowering of the systolic pressure. He attached much significance to these findings, and thought that the main factor was metabolic. It is perhaps a pity that this line of inquiry appears to have been dropped.—I am, etc.,

Helensburgh, Dumbarton. O. H. MAVOR.

SIR,—In their researches into the natural history of asthma Drs. Elsie O. Williams and G. E. O. Williams (October 22, p. 897) have presented a number of points and conclusions which lend support to the findings of other investigators, but at the same time have formed conclusions which might be regarded as highly controversial. In particular I would like to refer to the "relevance of hormonal allergy in women."

Few will dispute the increased incidence of asthma at puberty, and it may be significant that many become asthmatic for the first time at that period. In the same way the condition may appear initially or become aggravated during pregnancy. But, as stated in your annotation (October 22, p. 921), many are relieved of their distress when pregnant—a fact which is difficult to reconcile with the authors' theory. Important in this connexion is the finding of Kapeller-Adler[1] that the histamine values in the blood cells of pregnant women were comparable in amount with those of non-pregnant women. That positive skin reactions to endocrine hormones have been obtained is neither surprising nor convincing seeing that the results of skin testing are to be considered suspect rather than unequivocal evidence of allergic sensitivity.

Is not the reason for the greater frequency at puberty and in some pregnancies to be found, as has been suggested in the past, in the alteration of the mode of life of the adult woman as compared with the adult man? For at that time woman adopts a much more sedentary type of existence, with consequent slowing of metabolism and reduction of the stimulating effects of weather—influences which tend to encourage parasympathetic preponderances. The relative infrequency of asthma in the more primitive races supports this point of view. Certainly the encouragement in these girls of a more spartan type of existence is in itself frequently sufficient to ameliorate the illness to a very considerable degree. The question arises once again whether there is some intimate relationship between the increase of allergy and the increasing artificiality of our existence.—I am, etc.,

Glasgow. GEORGE KRASNER.

REFERENCE

1 *Lancet*, 1949, **2**, 745.

"Mantoux-conversion"

SIR,—There is a place in the English language for a term to indicate a change from the tuberculin-negative state to the tuberculin-positive state, but the use of two different terms with this meaning leads to confusion. In the American and English literature the term "Mantoux-conversion" is used to indicate this change, but some Scandinavian writers have preferred the term "Mantoux-inversion," and recently one English writer has followed them.[1] To the ordinary English reader "conversion" clearly indicates a change from a first state to a newly acquired one, while "reversion" equally clearly indicates a change from an acquired state to a pre-existing one. "Inversion" suggests merely a "turning upside down," with no implication as to the nature of the change.

For the sake of clarity and uniformity, therefore, it would be preferable for all writers on tuberculosis to use "Mantoux-conversion" to indicate the change from the tuberculin-negative to the tuberculin-positive state, as shown by the Mantoux technique. Mantoux-reversion is the correct term to indicate the change from tuberculin-positive to tuberculin-negative. I am sure that our Scandinavian friends, who have such a remarkable and enviable command of our language, will be willing to drop the term "Mantoux-inversion," which has no real etymological justification for use in the sense it has acquired.—I am, etc.,

London, W.1. CHARLES NEWMAN,
Chairman, Prophit Tuberculosis Survey Committee, R.C.P.

REFERENCE

1 Irvine, K. N., *B.C.G. Vaccination in Theory and Practice*, 1949, Oxford.

Myxoedematous Madness

SIR,—Surely it is time that the science of semantics and the art of logical reasoning were taught in our medical schools. I can read no alteration of opinion in Dr. Richard Asher's letter (October 8, p. 818), yet Dr. E. B. Davies (October 29, p. 982) writes as if such an alteration had been expressed. This is not scientific discussion but misrepresentation, which is fortunately not common in science.

"Mad" is a term used by the laity for behaviour which varies from the mean so far as to endanger the subject or his fellows; it excludes delirium, in which the patient appears physically ill. Dr. Asher used the term correctly in the title of his article, most of his patients being referred to hospital by an authorized officer—a layman—because their behaviour was grossly abnormal and they were not delirious. Dr. Asher's task was to assess the degree of abnormality, and, if his observations confirmed those of the authorized officer, to discover whether there was physical disease associated with the disordered behaviour. He was successful, and restored nine patients to physical health and normal behaviour. The relation between physical disease and abnormal behaviour appears to be that of cause and effect, for reasons explained in detail by Dr. Asher; they are perfectly logical reasons and suffice to establish a reasonable presumption that this hypothesis is true.

The term psychotic is being used in two senses: first, as equivalent to "mad"—as in the term "organic psychosis"; and second, as a technical term used in psychology. The first use includes states varying from "clouding of consciousness"

to catatonia, but the second is much more selective: many psychotic patients can earn a good living.

"Mad" patients with gross disorders of behaviour may be suffering from organic disease or from some disorder without discoverable organic cause. Most people know that an acute psychosis can develop apparently as a response to a severe emotional stress, but it is equally important to recognize that madness may result from organic disease. G.P.I., myxoedema, or epileptic equivalents may all appear in the observation ward, and, as far as I know, the response to treatment of the organic disease is at least as satisfactory as is that to psychotherapy.—I am, etc.,

Teddington, Middlesex. GEORGE DISCOMBE.

SIR,—In the interesting article by Dr. R. Asher on myxoedematous madness (September 10, p. 555) and in the annotation (p. 587) the often missed diagnosis of hypothyroidism is pointed out. In the list of signs that help the practitioner to establish a right diagnosis the presence of myoidema is omitted. However, in my experience the presence of a marked myoidema after mechanical stimulation of skeletal muscle is of significant practical value, since its absence virtually eliminates the diagnosis of hypothyroidism.

The localized muscular contraction that determines the erroneously called myoidema is observed in several conditions, generally accompanying a negative balance of nitrogen, but never reaches the proportions and specially never lasts so long as in myxoedema. Furthermore, the disappearance of the phenomenon after thyroid treatment indicates the direct relation between the abnormal muscular response and the endocrine disturbance. On the other hand, the rare and fleeting myoidema that is observed in hyperthyroidism is another evidence of the effect of thyroid gland upon the reaction of skeletal muscle, since in this condition, with its muscle wasting, a marked myoidema would be logical to assume.

This important sign was first observed in hypothyroidism and myxoedema by Södebergh,[1] who gave a right explanation of it. Recently, apropos of a study of skeletal muscle in hypothyroid conditions, I insisted on the practical value of this sign, which goes hand in hand with the results of the treatment.[2] It is objective and does not depend on the good will of the patient. It should be remembered that in patients with marked infiltration of subcutaneous tissue it is better to palpate the biceps than to look at it after vigorous pinching of the muscle, the better form of applying a mechanical stimulation. In other rare conditions not entirely due to a deficiency of the thyroid gland but undoubtedly connected with it—such as hypertrophia musculorum vera and the syndrome of Debré-Semelaigne—myoidema assumes enormous proportions.—I am, etc.,

Buenos Aires. ALFREDO LANARI.

REFERENCES

1 *Rev. Neurol.* 1910, **2**, 487.
2 *Medicina, B. Aires*, 1947, **7**, 197.

SIR,—I have always been struck by the way that opposing correspondents misunderstand each other's views. The correspondence about my article on myxoedematous madness (September 10, p. 555) seems no exception. Dr. E. B. Davies (October 29, p. 982) is pleased that I no longer hold my "unrestrained opinions," but in my letter (October 8, p. 818) I did not alter them, I only explained them again. I regard myxoedema as an important cause of insanity because it is important for people to know about it, because it is important to recognize and treat it, and because it is not rare and may often be rapidly cured.

I cannot quite understand why Dr. H. Bourne (October 29, p. 982) wishes to discount six of my cases because they had on occasion clouded consciousness. Their madness was due to the myxoedema just the same, and it certainly did not (as Dr. Bourne says) "respond to thyroid as they regained consciousness." Case 5 became so irrational that she upset the whole of the ward, and Case 10 became maniacal and hallucinated for over a fortnight (this is made quite clear in my paper). Of the three which he says failed to respond, two showed considerable improvement (again quite clear in the paper).

Dr. Bourne says it requires more evidence to show that myxoedematous madness is a more distinct entity than "rheumatic madness," "acute haemorrhagic madness," etc. The evidence (apart from these 14 cases) is to be found on page 31 of the report by the Clinical Society of London[1] and in all the other references given at the end of my article, whereas there is nothing known about rheumatic madness or acute haemorrhagic madness. Further evidence is supplied by letters and messages I have had from doctors who have met similar mental changes with myxoedema and also by two more cases I have seen since I wrote the article.—I am, etc.,

London, W.1. RICHARD ASHER.

REFERENCE

1 *Trans. clin. Soc.*, 1888, Supplement to vol. 21.

*** This correspondence is now closed.—ED., *B.M.J.*

Splenic Puncture

SIR,—Though sternal puncture has become an indispensable procedure in the study of the blood cells, in temperate zones diagnostic puncture of the spleen is becoming more frequently used. Splenic puncture is particularly helpful in the study of the cells of the lymphatic and reticulo-endothelial series and in the diagnosis of the obscure leukaemias and reticuloses. In the Tropics spleen puncture is much more frequently performed, especially for the diagnosis of kala-azar. Recently, however, splenic puncture has tended to become replaced by sternal puncture because of the supposed danger of the former and because in kala-azar the latter is supposed to give as many positive smears.

There is little justification for either of these suppositions. Although occasional deaths are reported as the result of spleen puncture, those with the greatest experience of the procedure find it perfectly safe.[1-3] Debono[4] in his experience of 2,000 cases of infantile kala-azar had only two deaths—one in a case of leukaemia and one in a moribund patient. Chung[5] reported 50 cases of kala-azar in which spleen smears gave fewer positives than either lymphatic gland or sternal smears, but in recent years he has seen cases in which sternal smears were negative and spleen smears positive (personal communication). Napier[6] in one series of 55 cases found 9 positive spleen smears in 11 cases with negative sternal smears. In another series[2] he found 9 out of 80 cases to have negative sternal smears; six of these nine patients were examined by spleen puncture, and positive smears were obtained in three. The writer has never found parasites in sternal smears when they were absent from spleen smears. The only large-scale comparative study is that of Ho and his colleagues,[7] who examined smears from both spleen and sternal punctures in each of 450 cases of kala-azar. They found sternal smears positive in 85.8%, while spleen smears showed leishmania in 97.6%. Napier and Ho agree that when parasites are present in both sternal and spleen smears they are easier to find in the latter.

During the past 18 months the writer has been using a simplified technique of spleen puncture the safety of which is demonstrated by the absence of mishaps in nearly 1,500 punctures, one-third of these being on cases of splenomegaly from causes other than kala-azar. The advantages of the technique are not only its safety and simplicity but also its rapidity and the fact that it can be carried out in every case regardless of the presence of blood changes. It requires no co-operation from the patient and can be used in infants and children, crying, struggling, or otherwise. Patients need no preparation or local anaesthetic, and they get up immediately after the puncture. The procedure may be repeated as necessary. The technique is as follows.

A dry all-glass 10-ml. syringe is used with a 22-gauge needle (1½ in. = 3.1 cm.). The plunger of the syringe must be well fitting but freely movable within the barrel, and with the needle attached the syringe should be tested for these points just prior to performing the operation. The patient lies flat on a bed, and the skin over the spleen is cleaned with alcohol and allowed to dry. The operator stands on the patient's right side and with his left hand palpates the edge of the spleen. The right hand inserts the needle under the skin about midway between spleen edge and costal margin. The left hand then takes the barrel of the syringe and the right thumb and forefinger take the head of the plunger. With a rapid movement of the left hand the needle is thrust for a depth of about 1 cm.

into the spleen and immediately withdrawn from the patient. At the same time that the left hand pushes the needle into the spleen the right hand forcibly withdraws the plunger, so that during the fraction of the second that the needle point is within the spleen a partial vacuum is produced within the barrel. (The needle penetrates the splenic capsule at an angle of about 75° to its surface, though, since there is no time for the spleen to move while the needle is within its substance, this angle is unimportant.) Enough material is obtained on the tip of the nozzle of the syringe or within the needle for the making of a smear.

A similar technique has been used for liver puncture and for puncture of spleens which are not palpable. In the latter case the position of the organ is determined by percussion and the needle thrust between the ribs. Before using this technique, the rapid, co-ordinated movements of the hands should be practised.—I am, etc.,

Brighton. EMANUEL TUCKMAN,
Director, Anti-kala-azar Campaign,
Friends' Service Unit (China).

REFERENCES

1 Henderson, L. H., *Trans. R. Soc. trop. Med. Hyg.*, 1938, **31**, 179.
2 Napier, L. E., *Principles and Practice of Tropical Medicine*, 1946, New York.
3 Shortt, H. E., *Trop. Dis. Bull.*, 1947, **44**, 145.
4 *Proc. R. Soc. Med.*, 1947, **40**, 155.
5 *Trans. Far Eastern Ass. trop. Med.*, 1938, **2**, 111.
6 *Lancet*, 1939, **1**, 959.
7 *Trans. R. Soc. trop. Med. Hyg.*, 1948, **41**, 629.

Lymphadenopathy in Brucellosis

SIR,—Dr. Hugh Conway's interesting article on lymphadenopathy in brucellosis (October 8, p. 787) was of great personal interest to me in view of the extensive work on the subject of Malta fever carried out by my father, the late Colonel J. C. Kennedy. He served in Malta from 1901–6, and was appointed a member of the Malta Fever Commission on its formation in 1904. In 1908 he received the Edinburgh University Gold Medal for his M.D. thesis on Malta fever. Parts I and III of this thesis deal with the aetiology, epidemiology, immunity, and prophylaxis of Malta fever, while part II is a description of the clinical conditions based on the personal observation of 250 cases. The following paraphrased extracts from this thesis seem to have some bearing on the question whether or not lymphadenopathy occurring in a patient with brucellosis is a specific manifestation of the disease.

1. In the section dealing with aetiology and experimental pathology the following findings are recorded. The experimental inoculation of *M. melitensis* into a monkey and two dogs caused enlargement of the spleen and certain lymph glands 20–60 days after the inoculation. *M. melitensis* was recovered (in profusion) from the mesenteric, femoral and axillary glands in the case of the monkey, and from the inguinal and axillary glands in the case of the two dogs.

2. Under the description of symptoms by systems is the following paragraph. "All the lymphatic glands are slightly enlarged, and those lying in the neck posterior to the sternomastoid muscle become tender very early in the disease. The glands are the last organs on the body for the micrococcus to leave (see morbid anatomy). Lymphangitis has occurred in one case in both legs with enlargement of the popliteal glands."

3. The following paragraph occurs under the heading "Post-mortem Bacteriology." "The organism *M. melitensis*, is recovered from the following organs in the greatest profusion in order of precedence: lymphatic glands (when fluctuating), spleen, liver, bone marrow, kidney. The observation that the lymphatic glands were so favourable to the recovery of the organism (Kennedy, *Malta Fever Reports*, IV) was of the greatest value from an experimental point of view, as it was proved that the organism remains in these organs long after it has disappeared from all other parts of the body. Therefore it became routine practice when making post-mortem examinations on experimental animals to make cultures from several of the glands. In many cases it was the saving of the experiment when these organs were the only ones containing the micrococcus."

In 1906, as a result of the findings of the Malta Fever Commission, the consumption of unpasteurized goats' milk was prohibited to all British Service personnel. Prior to that date the average incidence of Malta fever in the Malta garrison alone was in the region of 600 cases yearly. Since 1906 the incidence of Malta fever in the garrison has for the most part been only sporadic. For this reason it is probable that there will never again be such a wealth of material available for investigation and analysis. It is on these grounds that I submit the above extracts.

It may interest your readers to know that it was Colonel J. C. Kennedy who in January, 1914, provided the first clue that cows' milk might produce undulant fever in humans when he reported the presence of agglutinins for the *Micrococcus melitensis* in the milk and blood serum of cows in London.[1] Unfortunately, due to being posted overseas, he was unable to follow up this observation. It was four years later, in 1918, that Miss Alice Evans showed that these agglutinins found in the milk and blood serum of cows were due to infection by the closely allied *Brucella abortus*.—I am, etc.,

Penarth, Glam. M. C. S. KENNEDY.

REFERENCE

1 *J. R. Army med. Cps*, 1914, **22**, 9.

Tetraethylthiuramdisulphide in the Treatment of Alcoholics

SIR,—I am glad that Dr. J. N. P. Moore (October 22, p. 932) has drawn attention to the need for a careful technique in the exhibition of this new drug. In answering the many inquiries I have had regarding it I have never failed to stress the importance of a proper selection of patients and control of conditions. Dr. Moore does not, I think, realize that no patients are accepted at Caldecote unless they promise, with their lips at any rate, to co-operate whole-heartedly during whatever course of treatment is prescribed for them. They are warned that any surreptitious consumption of alcohol while they are under treatment and on parole is liable to bring upon them distress up to the point of danger. Actually I never have encountered a dangerous state, though laymen, seeing a man as red as a turkey-cock and in the throes of acute anxiety, have told me with alarm that he seemed to be *in extremis* and "about to pass out." Some patients to whom I do not wish to give the drug I invite to act as umpires while a more robust fellow is undergoing test. They finally conduct the latter to bed and declare that a personal experience is not necessary for them; the vicarious atonement suffices. Quite possibly there exist individuals allergic to tetraethylthiuramdisulphide just as there are those allergic to other substances—e.g., strawberries, belladonna, bee-stings, etc.—but such have not crossed my path, or I intuitively have passed them over. Dr. Moore does not refer to the interesting fact that if a resistant patient on a small regular dose of the drug manages also to take a small quantity of alcohol he develops as the days go by, in addition to a pink complexion shortly after drinking, peripheral neuritis and mild general confusion. I have known several such cases and deliberately let them continue in order to observe results.

As to giving the drug for a few months only—this is just to help a patient by affording initial confidence and a sense of security while he is facing life again after a course of general and psychotherapeutic treatment. The drug alone cannot effect a cure; it only can act as a useful ancillary to a psychological reorientation of the character defect which in the first instance induced the individual to try to bolster himself up by recourse to alcohol. No drug of itself ever cures addiction. Unless the personality is readjusted relapse ensues when and if, sooner or later, fate turns on the screw appropriate to elicit that individual's malreaction, whatever it be.

The value of your space precludes me from entering into a more detailed exposition of this thesis. I conclude with the caution that to exploit any drug, new or old, with the promise that it will suffice to make a person behave himself for what politicians call "all time" is worthy of condemnation as quackery, be it consciously or ignorantly pursued.—I am, etc.,

Nuneaton. A. E. CARVER.

The Artificial Kidney

SIR,—With reference to letters and articles on the renal shunt and the artificial kidney I beg to suggest a much less drastic procedure to deal with anuria. In 1906 and 1907 I performed my first experiments with diathermy at the Veterinary Institute of the University of Berlin. After exposing by laparotomy both kidneys of rabbits or dogs and then treating one kidney from the back through all the integuments I noticed that the treated kidney assumed after a minute or two a highly arterial coloration while the other kidney remained bluish. I explained this effect by the local spasmolytic action of diathermy and

consequently used it as a life-saving device in cases of anuria. Not only was the severe pain in the back relieved in every case, but a subsequent flood of urine took place within one to several hours. The treatment should be of 30 minutes' duration, with maximal bearable intensity short of blistering, and should be repeated hourly until the flood of urine starts. In cases of not purely reflex anuria, which may be due partly to mechanical obstructions and partly to their reflex action, considerable amounts of sediment may appear in the first portions of urine.

Before deciding to use the artificial kidney, this very simple method should be tried. It is essential, however, to use classical and not short-wave diathermy, because of its specific action on the vascular nervous supply, as I have explained in my text-book on clinical diathermy which I hope will be published in the near future.—I am, etc.,

Manchester. K. F. NAGELSCHMIDT.

Poliomyelitis Vaccination

SIR,—The difficulty of immunizing against poliomyelitis as your correspondent, Mr. M. H. F. Johnson, suggests (October 1, p. 757) is that, apart from considerations of the safety and efficacy of the vaccine, the majority of strains of virus can only be propagated in monkeys, supplies of which are totally inadequate for any large-scale production of vaccine. If he wishes to know more on the question he might read the article by W. McD. Hammon on immunity in poliomyelitis in *Bacteriological Reviews* (September, 1949, **13**, 135).—I am, etc.,

Birmingham. B. R. SANDIFORD.

Aetiology of Erythema Nodosum

SIR,—Dr. S. A. Doxiadis, in his article on the aetiology of erythema nodosum (October 15, p. 844), stressed the importance of erythema nodosum as a signal of primary tuberculosis infection in children. My experience, based on 2,124 cases of childhood tuberculosis seen in the Dublin Corporation's primary tuberculosis clinic, may be of interest. This clinic was established in 1945, and the material is obtained from tuberculosis clinics, children's hospitals, and general practitioners. I have found only 61 cases of erythema nodosum in the total of 2,124 children—i.e., 2.8%. These 61 cases were all tuberculin-positive.

I would be interested to know if the incidence of erythema nodosum is greater in Dr. Doxiadis's material than has occurred in our case material in Dublin. Could Dr. Doxiadis give any estimate of the number of primary infections which had to be reviewed to produce the 100 cases of erythema nodosum published in his instructive article?

Compared with Scandinavian figures, our incidence of erythema nodosum is remarkably low. Every case was carefully questioned on this point in case the parents considered this manifestation of primary tuberculosis too trivial or did not connect it with the child's illness. My view, based on experience in an Irish sanatorium, is that erythema nodosum, as a sign of primary tuberculosis, occurs in the older age groups. Probationer nurses often show their primary infection by this sign. In Norway the age distribution curve for erythema nodosum presents a sharp maximum in the 20–25 age group. In Scandinavian countries primary infection occurs mostly in young adults, and childhood infection is rare. In Ireland childhood infection is common. As erythema nodosum is principally an affection of adolescence, this may account for the high incidence of this sign in Scandinavia (30%) and its low incidence in childhood primary tuberculosis here (2.8%). Eight of this group of 61 were under 5 years of age—all the others were in the 5–14 age group. Following the usual trend of this allergic reaction, 39 cases occurred in girls and 22 in boys.

The time interval between this outward sign and radiological evidence of primary infection was studied. I have observed that the erythema nodosum often preceded the *x*-ray evidence by one to three months; but in two cases pleural effusion showed, one six months later and one a year afterwards. I think it is of great importance that these cases should have repeat *x*-ray films, since films, including laterals, taken only on one occasion may miss the lesion. Some of our cases had *x*-ray evidence when the erythema was noticed, but two other cases which were tuberculin-positive showed no radiological evidence, although each had three *x*-ray examinations at intervals. Another interesting case had two attacks of erythema nodosum, the primary infection being diagnosed at the second manifestation. This last case, in a child aged 10 years, had a history of erythema nodosum diagnosed at a city hospital in April, 1944. She had a second attack when seen at the primary clinic in March, 1945. Her sputum was then positive; B.S.R. was 17 mm. in one hour, and there was cavitation seen on *x*-ray examination. She died in a sanatorium in February, 1948. This was the only death in the erythema nodosum group.

Perhaps Dr. Doxiadis can confirm my observations that erythema nodosum often precedes the radiological evidence of primary tuberculosis infection in children.—I am, etc.,

Dublin. M. DUNLEVY.

The Acute Throat

SIR,—In Dr. T. Anderson's very thorough article on the acute throat (October 15, p. 860) he states, referring to penicillin: "Local therapy by lozenges and so forth is therefore completely inadequate and systemic administration in high dosage is indicated." He then goes on to state that injections of the soluble compound four-hourly, or the insoluble procaine penicillin two or three times a day and later less frequently, are indicated for seven days. Later, referring to sulphonamides, he remarks, "The efficacy of these drugs has never been convincingly demonstrated in such conditions."

I feel I must disagree. One sees all varieties from mild to severe cases of tonsillitis (mostly caused by the haemolytic streptococcus): the mild cases nearly all clear up with penicillin lozenges and possibly an aspirin mixture, and the severe nearly all clear up with lozenges aided by sulphonamides by mouth and nearly, if not all, in two to five days. Very few G.P.s have the time to give all the recommended injections; the district nurses cannot possibly afford the time; the hospitals are too full; and most patients to-day cannot afford private nurses or nursing-homes.—I am, etc.,

Hove, Sussex. W. A. PORTER.

Sex Hormone Upset in Africans

SIR,—Dr. J. N. P. Davies's paper (September 24, p. 676) on this subject offers a hypothesis which would, if confirmed, throw a good deal of light on some of the perplexities of Bantu medicine. Being at present engaged on a clinical study of malnutrition in Zulu children, we would wish to comment on one or two points raised by Dr. Davies, especially where our own experience differs from his findings further north.

Trowell's findings regarding the prevalence and nature of the syndrome of kwashiorkor (malignant malnutrition) are amply in accordance with the situation in Zululand, but the suggestion made in the paper that few African children escape without at least a mild attack owing to the prolonged period of breast-feeding and the absence of a proper weaning period appears contrary to our findings. Traditionally the breast is supplemented from birth by sour maize porridge (*p*H 4–6), and breast-feeding (other than the mere act of dangling the child on its grandmother's shrunken breast) is usually discontinued by the end of a year, the child then being fed entirely on maize porridge with a little sour milk if available. Weaning may therefore be said to be going on all the time and to be extremely gradual.

Further, we have been able to induce a considerable number of mothers to feed wholly on the breast for six months and then to wean over the next three or four months, using maize products and milk. It would not appear so much that prolonged breast-feeding and the absence of a proper weaning period were at fault as what a child is fed on during and after weaning. Malignant malnutrition is common in children who have been weaned slowly and breast-fed for a "normal" length of time. Preliminary studies have shown that children wholly breast-fed to six months teeth two to three months earlier than their fellows who are fed on the traditional breast-and-porridge

routine. We hope to publish detailed studies at a later date on this matter.

Infertility does not seem so great a problem in Zululand as Dr. Davies describes in Uganda. Of 243 women (a small figure and part of an uncompleted series) past childbearing age, eleven (4.5%) had had no children, while 74% had four or more children.

While umbilical hernia is extremely common in Zulus (a great many of these hernias appear to undergo spontaneous regression about puberty), inguinal hernia appears relatively rare. In 5,000 admissions we recorded only six cases, but it must be remembered that a majority of males are away from this area for many months each year, working in the mines and industry.—We are, etc.,

E. A. Barker.
Margaret Barker.

Nqutu, Zululand.

Significance of Convulsions in Infancy

Sir,—The correspondence on convulsions in infancy (September 3, p. 547, and September 10, p. 598) raises the question whether hyperpyretic convulsions are necessarily the forerunners of epilepsy in later life. This is still a controversial issue.

On the one hand, Peterman[1] maintains pessimistically that every convulsion in a child, unless it is caused by encephalitis, meningitis, or poisoning, has to be considered as the first manifestation of a later epilepsy. In his series of 55 cases of convulsions associated with fever an electroencephalogram was done in 34; only five had normal tracings, and in 35 the diagnosis of epilepsy had to be given subsequently. On the other hand, Neyroud[2] is more optimistic: he states that in the majority the hyperpyretic convulsions may be considered as a common accident which has nothing to do with epilepsy and the prognosis of which is favourable. In his series of 70 children the electroencephalogram was normal in 57, doubtful in nine, definitely abnormal in four, and two showed epileptic patterns.

These extremes will have to be reconciled by further research. Apparently some children will have convulsions when a certain set of circumstances is favourable for their initiation—e.g., fever, with its hyperventilation, and changes in the water metabolism. These conditions may never be imitated later on in life and no epilepsy will ensue.

The therapeutic question, in my opinion, boils down to this: there is no need to start anticonvulsant treatment in every disclosed dysrhythmia so long as it does not give rise to symptoms such as manifest epilepsy, behaviour disorders, or irritability. In febrile convulsions, when no electroencephalogram is available for the elucidation of the case—and at present it is not for a good many general practitioners—the exhibition of antipyretics and phenobarbitone whenever a febrile illness makes its appearance will be all that is necessary. This seems to me a better scheme than that suggested by Dr. G. Daynes (p. 547), who advises anticonvulsant drugs "during the years when the threshold is low." There is no knowing when to discontinue and also a risk in a case of idiopathic epilepsy of status epilepticus after withdrawal of prolonged anticonvulsant treatment.—I am, etc.,

E. Guthaner.

Gatton, Queensland.

References

1 *J. Amer. med. Ass.*, 1948, **138**, 1012.
2 *Praxis*, 1947, **36**, 759.

Treatment of Basal-cell Carcinoma

Sir,—As a dermatologist who over a period of years has treated a very large number of rodent ulcers with radiation, I would like to draw attention to one point in connexion with their aetiology which I feel should have some bearing on the selection of treatment. Among the cases I have treated there has been a number of "recurrences" in excision scars. Knowing who operated in most of these cases, I have not often entertained any suspicion that the lesions were not widely enough excised in the first place.

I think that most of us to-day recognize trauma as the cause of a considerable proportion of the cases of basal-cell carcinoma seen. Patients present themselves with a history of having had a scratch from a pin, a rose thorn, etc., or a cut with a razor blade, which healed but broke down to form a "sore" which would not clear up. In other cases the original injury never healed. In a person with a proclivity to develop rodent ulcers a surgical incision constitutes a greater skin injury than does a scratch with, say, a pin or a rose thorn. I believe that many of the so-called "recurrences" are not really recurrences at all. In my opinion a basal-cell carcinoma had developed in an excision scar in a person with a tendency to develop skin malignancies. Quite recently I saw two cases which illustrate this point.

The first was in an elderly woman who three years ago had a small microscopically proved squamous-celled carcinoma excised from the side of her neck because she could not spend enough time in the city to undergo a course of radiotherapy. She returned recently with two basal-cell carcinomata, one towards either side of the 3-in. (7.5-cm.) excision scar. She had keratoses and another rodent ulcer as well. The second case was that of a woman who had had a mastectomy done some years ago and who presented herself with two basal-cell carcinoma (proved by biopsy) in the mastectomy scar. She was a freckler and had keratoses on the hands and face as well.

I am bringing this point up because I feel that patients who show evidence of a tendency to develop malignant conditions of the skin, keratoses, pigmented patches, etc., are much more likely to develop new basal-cell carcinoma in excision scars than patients who show none of these stigmata. I feel it is much safer to treat them with radiation than by excision.—I am, etc.,

J. M. O'Donnell.

Perth, Western Australia.

Identification of Microfilaria

Sir,—I have for a long time been interested in getting a quick method, of value in field work, by which the various species of microfilaria could be identified. Lately I have used the brilliant-cresyl-blue method as a vital staining, and I have been surprised at the rapidity and efficiency of the method, which is as follows.

Slides are prepared by dropping on them a drop of a saturated alcoholic solution of brilliant cresyl blue. On the dried slides a drop of blood is placed. A cover slip is placed over the drop, the edges sealed off with vaseline, and the slide examined immediately. The microfilariae will be found to survive for about five minutes, and the granules will be distinctly stained, allowing easy identification.

—I am, etc.,

E. S. Monteiro.

Singapore.

Medical Services of the Armed Forces

Sir,—The scope of Major E. A. Smyth's article (October 1, p. 751) is so large that detailed comment on all its points is difficult. May I write on one or two he has not mentioned?

(1) Could not the smaller formations, such as field ambulances, have a non-medical adjutant for administration purposes? While fully admitting the necessity for medical officers in these units to be well versed in man management—which is inborn, not acquired—training, discipline, strategy, map-reading, etc., it always seemed to me a gross waste for medical men to be chained to a desk for hours every day, grappling with audit boards, imprest accounts, courts of inquiry, summaries of evidence, messing, rations, and pay, when they should have been out with their men, getting to know them, supervising training and exercises.

(2) Has, I wonder, any conference been called at the Staff College to which distinguished Territorial officers (who commanded field ambulances and casualty clearing stations by the dozen, and were A.D.M.S.s to at least four or five famous fighting divisions, D.D.M.S.s to at least two corps and to one Army command) have been invited? Could not their elasticity of mind and their genius for improvisation—the acquiring, for example, of thousands of extra square yards of canvas to supersede the miserable pent-houses on the three-tonners—be an asset? Could not their knowledge and experience be made use of in considering future wars? Perhaps this has been done. If not, it is surely overdue.—I am, etc.,

W. A. Bellamy.

Aylesbury, Bucks.

Paddington Green Children's Hospital

SIR,—Your readers are probably familiar with the decision of the board of governors of St. Mary's Hospital to convert Paddington Green Children's Hospital to adult use and for the reason which has been given for this action—namely, the excess of children's beds in the St. Mary's group over the number recommended in the Goodenough Report. They may also have read the Ministry of Health circular (R.H.B. (49) 132), which together with your annotation entitled "Changes in Use of Hospitals" (October 15, p. 857) encouraged many to believe that the board might reverse their decision. Unhappily, there is no sign of any change of heart.

I am therefore writing, as chairman of a committee which is composed mainly of members of the local public and which is non-political, to give publicity to this fact. It seems right that the profession should know exactly what is happening, because this is certainly no academic medical dispute. In Paddington and neighbouring boroughs a great wave of spontaneous public indignation has occurred and now finds expression through the Save the Children's Hospital Committee. Twenty thousand people, mostly local, have already signed a petition, and further evidence is found in the resolution passed by the Paddington Borough Council on November 1, after a special committee appointed by them had investigated the matter, deploring the decision of the St. Mary's board. It is apparent that the board of St. Mary's Hospital has failed completely to understand the depth of feeling which their action has aroused. Their explanation, that students must be properly taught, does not carry conviction. Hitherto there has been no suggestion that the doctors produced by St. Mary's are less capable than their colleagues.

My committee feels a need to work hard to bring about the final decision in the near future, since a hospital which is under threat of annihilation works under strain, and it is only a matter of time before signs of deterioration must appear through the unwillingness of serious workers—whether doctors, nurses, or others—to spend their energy on tasks which have no relation to a future.

Surely the proper course is to allow the Children's Hospital to continue the work which it has carried on with acknowledged success for 66 years, and to allow its teaching facilities to be transferred to another teaching hospital group less fortunately situated than St. Mary's.—I am, etc.,

MICHAEL HARMER,
Chairman, Save the Children's Hospital Committee.

London, N.W.1.

The G.P. and E.C.T.

SIR,—The experience mentioned by Dr. Donald M. O'Connor under the above heading (October 29, p. 984) has been unfortunate, but it may have been due in some instances to factors other than mere inefficacy of E.C.T. Many of our modern techniques (E.C.T., insulin coma, prolonged narcosis, leucotomy, etc.) do not so much cure the patient as render him more accessible and responsive. In other words they pave the way for other therapeutic methods, and if these are not applied the patient remains half-treated and incompletely recovered.

Some cases, after special physical treatments, require psychotherapeutic help, while many need weeks, even months, of intensive rehabilitation under good hospital conditions. Whether this takes the form of occupational therapy, physical training, and special games for one patient, of outdoor sport, general entertainments and competitive outlets for another, or of cultural activities, social functions, and club conditions for yet another, such rehabilitation is unlikely to be available except to in-patients, and it is in hospital that proper combinations of all the required activities can best be prescribed, organized, and carried out.

It is unfortunate that there has been a temptation to hail the newer physical treatments not only as applicable to too wide a range of conditions but also as complete and sufficient therapies in themselves, forgetting that mental stability and a resumption of normal life cannot always be achieved without the application of rehabilitating and resocializing methods before the patient returns to his home, so that if he leaves hospital without adequate treatment during his convalescence the risks of a relapse will naturally be all the greater.—I am, etc.,

Warrington, Lancs. J. ERNEST NICOLE.

Catalogue of Medical Films

SIR,—Two years ago, with the aid of a generous grant from the Royal Society of Medicine, the medical committee of the Scientific Film Association set up a "master file" of data on medical films. It includes details of production, distribution, and content, and a provisional grading of each film. It covers all the films in existence in this country up to 1946 and is being added to constantly. Covering over 800 films, it provides the most comprehensive collection of data about its medical films that any country in the world (with the possible exception of Canada) can show. From this master file was prepared the RSM-SFA catalogue of medical films. The first edition of 1,000 copies was exhausted in twelve months, a remarkable index to the widespread interest in medical films.

We are faced with the problem of deciding whether to reprint or to prepare a new edition. As the lay-out of the first edition was cramped by paper shortage, and as there are many new films to be included, we have decided in favour of a new edition. We are constantly getting requests from abroad as well as from this country, and we ask the hospitality of your columns for this explanation of why the catalogue will not be available for some months yet.—I am, etc.,

4, Great Russell Street, London, E.C.1.

KENNETH GOADBY,
Chairman, Medical Committee, Scientific Film Association.

POINTS FROM LETTERS

Retinal Arterial Occlusion in Migraine

Dr. LEO HAHN (Leicester) writes: I have been most interested in Dr. G. S. Graveson's article (October 15, p. 838), which provides an important contribution in support of the time-honoured vascular theory of the migraine attack. As long ago as 1923 I accepted the view that the migraine paroxysm is initiated by a cerebral angiospasm, and introduced the potent vasodilator drug papaverine into the treatment of migraine (*Dtsch. Z. Nervenheilk.*, 1923, **77**, 191). Forty milligrammes of papaverine hydrochloride, if given intravenously—very slowly in order to prevent a fall in blood pressure—early in the attack, rarely fails to arrest the paroxysm. Administered orally in much higher doses (100 mg. t.d.s.) for long periods, the drug is in most cases effective in reducing the frequency of attacks. The effectiveness of this treatment has been confirmed by several authors. . . . Dr. Graveson's cases illustrate very well that occasionally the cerebral disturbances characteristic of the aura and caused by the initial angiospasm may substitute the attack. Together with Dr. E. Kraupa I have reported a case of spasm of the central retinal artery producing the picture of "acute retinal ischaemia" with subsequent amaurosis in a girl 5 years of age (*Klin. Mbl. Augenheilk.*, 1921, **66**, 829). Papaverine treatment restored within a few weeks the retinal circulation and the visual acuity. It seems probable that the early administration of papaverine in cases as reported by Dr. Graveson could prevent the thrombotic vascular occlusion.

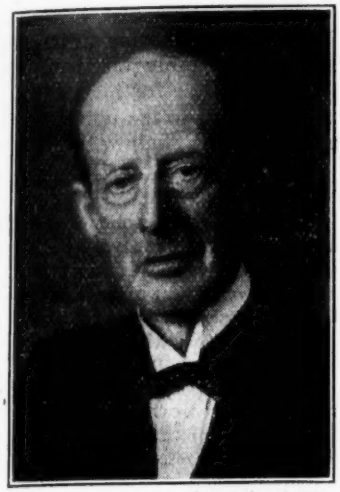
Migraine and Calcium Deficiency

Dr. N. M. STEPHEN (Saxmundham, Suffolk) writes: With reference to the article (October 15, p. 838) by Dr. G. S. Graveson calling attention to a condition of arterial spasm in migraine, I have the following observations to make. Acting on the assumption that migraine might be considered to be a vasomotor disorder involving changes in the vascular tone of the arteries of the meninges, I made an attempt in a series of severe cases of migraine to try the effects of calcium therapy. The form of calcium used was colloidal calcium with vitamin D. The results were surprisingly successful. Therefore I have concluded that migraine is due in part at least to a genuine calcium deficiency. The treatment was used empirically, and I had no facilities for carrying out estimations of the serum calcium in the patients treated. An interesting fact is that one of the patients stated that she had been free from attacks of migraine while she was pregnant. This might be explained by supposing that all supplies of calcium are mobilized during that period and are therefore more readily available. All the patients referred to had already experienced all the usual forms of treatment—i.e., aspirin, phenobarbitone, and ergotamine tablets. The time interval between the injections tends invariably to increase, and a space of two to three months may elapse before the next injection becomes necessary.

Obituary

Sir HERBERT EASON, C.B., C.M.G., M.D., M.S. F.R.C.S.

Sir Herbert Eason, president of the General Medical Council since 1939, died at Guy's Hospital on November 2 at the age of 75. He had attained distinction in the practice of a medical specialty, in hospital administration, in the government of a university, and as president of the body controlling the training and discipline of the medical profession. So closely associated was he with academic and public life that it was often forgotten that he was also an ophthalmic surgeon who had contributed much to the literature and teaching of ophthalmology. At Guy's Hospital he was chief of the ophthalmological department for more than thirty years. Herbert Lightfoot Eason was born in London on July 15, 1874, and received his education at University College. From there he went to Guy's medical school, qualifying in 1898, and taking his M.D. degree in 1901 and his M.S. a year later. In 1902 he became resident warden at Guy's, and in the following year dean of the medical school, a post he held until 1912. During the first European war he served with the rank of lieutenant-colonel as consulting ophthalmic surgeon to the Forces in the Mediterranean, Egypt, and Palestine. He was mentioned in dispatches, and awarded the C.M.G. in 1917 and the C.B. in 1918. His experiences in the Near East are recorded in *Guy's Hospital Reports* of that period and in the *Transactions* of the Ophthalmological Society, of which at that time he was an active member.



[Russell and Sons

Herbert Eason's appointment as superintendent of Guy's Hospital took place in 1920, when Sir Cooper Perry retired, and he made his home in the elegant old building, since destroyed by enemy action, on the east side of the famous court in which stands the statue of Thomas Guy. This post he held for not far short of twenty years, during which time many notable developments took place in the hospital. One of these was the opening by the Prince of Wales, afterwards Edward VIII, of Nuffield House for private patients, situated in the hospital grounds.

The affairs of the University of London, then in a transitional stage, began to claim his attention in 1911, when he was elected to the Senate as a representative of the Faculty of Medicine. He also represented the Senate itself on the University Court. Eason was chairman for various periods of the library and finance committees and of the advisory council, and in 1935 he became vice-chancellor. This office he held until 1937, when the Senate appointed him to the post of Principal of the University for a period of four years. The construction of the great university buildings in Bloomsbury, which during the war were the home of the Ministry of Information, had already been started, and Eason's appointment to the post of principal synchronized with the flying of the university flag from the completed tower. These four years were important ones in the history of the university. Eason had already been a member of the departmental committee of the Board of Education on the reconstitution of the university, and to a large extent under his guidance the reconstitution was effected and great progress made in all branches of its work. Research was promoted by scholarships and other means, and a large university professoriate was built up. At the end of his term of office as principal London University had 250 professors, 167 readers, and nearly a thousand recognized teachers.

Eason's outstanding work for the university by no means exhausted his activities in the educational field. He was for six years a member of the departmental committee of the Ministry of Health on postgraduate medical education, and he served as a member of the governing body of the British Postgraduate Medical School, which, like the twelve undergraduate schools in London, is a school of the university. It was his association with the University of London which brought him on to the General Medical Council ; he took his seat as its representative in 1925. His already great administrative experience proved most useful in this field. He took a prominent part in the Council's discussions on education ; he also became co-treasurer of the Council and a trustee of the English Branch. In whatever activity he engaged he seemed to make an effortless progress towards the top, and when Sir Norman Walker retired in 1939 Eason was the unanimous choice of his colleagues as President. He brought to the chair a long business experience as well as a natural authority, wisdom, and urbanity. A fierce light often beats on the G.M.C., but Eason was neither flattered nor intimidated. Throughout his career he had the most jealous regard for the ethics of his profession. He carried this to such a point that in his various official positions he would not allow his name to appear in the newspapers if it could be avoided. When newspaper men called on him for information he would tell them that they might say that the superintendent of Guy's took such and such a view or that the principal of the University of London had this or that intention, but the name of Eason they must not print, and generally his wishes were respected.

In a lecture he once delivered to students at Guy's Hospital he said that the simple rule which should govern them in their professional career was that same unwritten rule which guided the true Englishman in all circumstances—namely, that certain things are not done. This rule, he said, extended from trivial affairs of taste right down to cheating or embezzlement. "Gentlemen," said Eason, "the code of conduct summed up in the words 'it is not done' begins in the public school and goes on through the university, and remains with a man throughout his career. Keep those words in mind, and you will never come before the tribunal over which at the moment I have the honour to preside."

Eason was a member of the General Nursing Council, on which he represented the Ministry of Health. He was a co-opted member of the Public Health Committee of the London County Council, a member of the General Council and of the Economy Committee of the King Edward's Hospital Fund for London, a J.P. of the County of London, and a trustee of the Beit memorial fellowships for medical research. He was an honorary Bencher of the Inner Temple, and he contributed the articles on ophthalmology to French's

Index of Differential Diagnosis. Like some others who have occupied leading positions on the General Medical Council, in order to remain unembarrassed in dealing with disciplinary cases he stood outside the membership of the B.M.A., but he was sympathetic to the Association's aims, and in earlier years he rendered it good service as a member of the Ophthalmic Committee which helped to sponsor the National Eye Service. His name appeared as a knight in the birthday honours list of 1943.

RICHARD LAKE, F.R.C.S.

Mr. Richard Lake, who was for many years a well-known ear, nose, and throat surgeon practising in London, died in a nursing-home at Bristol on November 1 at the age of 87.

Richard Lake was born at Sittingbourne, Kent, on December 14, 1861, and was a student at St. Thomas's Hospital, where he qualified in 1885. In the same year he took part in the Sudan campaign as a surgeon of the National Aid Society. This was before the organization of the Red Cross, and he undertook similar work in the Servo-Bulgarian war, for which he was decorated by both the Servian and Bulgarian Governments. Returning to England, he took the F.R.C.S. in 1890, and from then onwards specialized in the treatment of diseases of the ear, nose, and throat.

Mr. Lake was appointed surgeon laryngologist to Mount Vernon Hospital in 1897, and in the following year began his long association with the Royal Ear Hospital. For many years he was consulting surgeon in his specialty to University College Hospital and to the Seamen's Hospital, Greenwich, and he was president of the otological section of the Royal Society of Medicine. During the 1914–18 war he served as aural surgeon to the R.F.C. Hospital and to Princess Christian's and Robert Lindsay's Hospitals for Officers. For four years from 1924 he was Geoffrey E. Duveen Lecturer on otology in the University of London. He was also at one time president of the West London Medico-Chirurgical Society. He published a number of papers on tuberculosis of the larynx, simulated deafness, aural vertigo, re-education of the deaf, mastoid suppuration, and other subjects in his specialty.

C. O. HAWTHORNE, M.D., F.R.C.P.

J. M. A. writes : May I pay tribute to the memory of my patient and friend, Dr. C. O. Hawthorne (October 29, p. 986) ? His great age brought increasing infirmity, but he never once complained. The disability which interfered with the use of his typewriter was a blow to him, and his friends missed his pungently witty notes. His command of the English language, verbal and written, was superb, and no man was more widely read. He was equally at home in the German language. When in 1942 his house in Hove was almost destroyed by a bomb he maintained an admirable philosophic calm. His interest in his profession and in the British Medical Association remained unabated to the end of his life. To the last his amazing memory was unimpaired, and there was no senile reiteration of old stories, but a gentle irony when discussing current events and modern medical politics. Only this year he published a popular science pamphlet, *The Human Body : A Justification.* Physically and mentally he was a big man, and beneath his reserved manner lay a vast understanding of human frailties. Although he outlived most of his contemporaries, he will be missed by all who knew him.

Dr. Bernard Myers writes : As a colleague of Dr. C. O. Hawthorne for many years at the Royal Waterloo Hospital I would like to mention the high respect in which Hawthorne was held by his colleagues for his downright honesty in his views, even if disagreeing with those expressed by the other members of the medical committee. He always spoke sound common sense and was fair in argument. If his external manner was apt to be severe, that was not the real man, for when we lunched together his genial and kindly spirit was evident. Those who knew him well will mourn the passing of a sound physician, loyal colleague, and good friend.

Dr. Eva McCall writes: May another old student of Dr. C. O. Hawthorne also pay a tribute to his memory ? Dr. Hawthorne lectured to the women students in the medical faculty at Queen Margaret College, University of Glasgow, in the 'nineties, and I was privileged to be one of those. He was a most inspiring teacher, and dressed the dry branches of materia medica with glowing tints, so much so that this dull subject became quite fascinating, and I owed him a lifelong debt of gratitude for his magnificent teaching. It was an incalculable loss to the university, where he would have filled a professorial chair with great distinction.

Dr. ARTHUR HILL BURNETT died suddenly on October 7 at the age of 68. He was born in Southampton and educated at King Edward VI School and the old Hartley College. He carried out his medical training at Edinburgh University and the London Hospital, qualifying in 1904. He held house appointments at the British Lying-in Hospital, Endell Street, at St. Mary's Hospital for Women and Children, Plaistow, and at the Kensington Infirmary, London. For a time he was a ship surgeon and visited many parts of the world. During the first world war he served in the R.A.M.C. with the rank of captain. In 1919 Dr. Burnett was appointed honorary anaesthetist to the Royal South Hants and Southampton Hospital, and medical officer to St. Mary's Institute. When the latter building was blitzed in 1940 he continued his work at Moorgreen Hospital. Apart from conducting a busy practice and his institutional work, he gave a large part of his time to the administration of anaesthetics at the Royal South Hants Hospital, where he was recognized as an expert anaesthetist. Retiring from this post during the recent war, he was appointed honorary consultant anaesthetist to the hospital. Dr. Burnett was an able physician, a skilled anaesthetist, and the pleasantest of colleagues. His contemporaries will long remember his happy disposition, his sincerity, and his great friendliness. To his widow and sons many colleagues and friends will extend their deep sympathy. —S. N. L.

Dr. CHARLES ROPER, of Rochester, who died there on October 17, was the younger son of the late Charles Roper, of South Kensington. He was born in 1877, and was educated at St. Paul's School, where he obtained a Tancred studentship in physic to Caius College, Cambridge, going into residence there in 1895. After taking the Natural Sciences Tripos he was awarded a scholarship to the medical school of the Westminster Hospital, and graduated B.C. in 1903 and M.B. in 1904. He proceeded M.D. in 1911, and took the Oxford D.P.H. in the same year. After filling house appointments at the Westminster Hospital he held for a few years an appointment as medical officer in Assam. Later, after his career had been interrupted by tuberculosis, he took up the study of that disease as a specialty. He was medical superintendent of Tehidy Sanatorium in Cornwall, and then tuberculosis officer for Kent, with headquarters at Rochester, and there he remained for the rest of his professional life. He had contributed on several occasions to the literature of his specialty. Dr. Roper was chairman of the Rochester, Chatham, and Gillingham Division of the British Medical Association from 1930 to 1932. He married Ethel, daughter of Mr. G. A. Dick, who survives him.

Medico-Legal

DANGEROUS DRUGS IN LOCKED CAR

A doctor was recently fined for keeping dangerous drugs other than in a locked receptacle.[1] His appeal, by way of case stated by Lord Ilkeston, the stipendiary magistrate for Birmingham, was heard in the King's Bench Division on October 19. Information against the doctor was originally preferred by a chief inspector of police, who alleged that on January 9, 1949, the doctor had contravened regulation 5 (2) of the Dangerous Drugs Regulations, 1937, which provided that "every drug . . . in the actual custody of a person authorized by this regulation shall, except when the necessities of the practice of the profession . . . by virtue of which that person is authorized . . . require, be kept in a locked receptacle which can be opened only by him or by some other person authorized by virtue of this regulation."

The facts proved before the magistrate were as follows: The doctor, as a registered medical practitioner, was a person legally authorized to be in possession of drugs to which the Dangerous Drugs Act, 1920, and the regulations made under it applied. On January 9 the doctor went to the cinema in his car, which he left in a car park with the doors locked. Inside the car was an unlocked leather case containing a number of dangerous drugs. On leaving the cinema he found that the car had been stolen. It was recovered on January 12, unattended and with all the doors wide open and the unlocked leather case containing the drugs on the rear seat. The drugs were handed to a police inspector, who later saw the doctor. The doctor admitted that the drugs were his.

It had been contended for the prosecution that a locked car was not "a locked receptacle" within the meaning of regulation 5 (2), and that if the doctor wished to take advantage of the exception in the sub-regulation the burden of proving the necessary facts for that purpose was on him. For the defence it was held that the doctor had complied with the regulation by leaving the drugs in an unlocked case in a car all the doors of which were locked. The key to the car was in the doctor's possession and was peculiar to that car. The word "receptacle" was not limited to the container of the drugs; the real test was whether the drugs were safely under lock and key. It was also argued that it was essential for the doctor to have these drugs easily accessible in case of emergency, and that it was much safer for him to leave the drugs in an unlocked case in a locked car than to leave them in a locked case in an unlocked car. The magistrate found the charge proved, and the doctor was therefore fined.

Judgment

The Lord Chief Justice, giving judgment,[2] said that in his opinion the regulation contemplated that drugs of this kind should be permanently and regularly kept in some locked receptacle. It was impossible to say that a motor-car was a "receptacle" within the meaning of the regulation. If it were, it could be held that if a doctor left dangerous drugs in his house and then went out and locked all the doors the drugs had similarly been left in a locked receptacle within the meaning of the regulation. A motor-car might be described as a receptacle for luggage, but the regulation must be construed so as to effect the obvious purpose of the Legislature. A motor-car which had constantly to be locked or unlocked, according as it was being driven or parked, could not fairly be brought within the terms of the regulation as being a "receptacle." The appeal was therefore dismissed.

[1] *British Medical Journal*, October 8, 1949, p. 819.
[2] *The Times*, October 20, 1949.

According to a report issued by the International Tuberculosis Campaign about 16,000,000 children and young adults in the countries in which Unicef is conducting feeding programmes had been tuberculin-tested and about 7,500,000 had been vaccinated with B.C.G. up to August 1, 1949.

Medical Notes in Parliament

NATIONAL HEALTH SERVICE (AMENDMENT) BILL

Lord SHEPHERD in the House of Lords on November 1 moved the Second Reading of the National Health Service (Amendment) Bill, which had already passed the House of Commons (*Journal*, October 29, p. 989). He described the Bill as small and non-contentious. In certain particulars it applied also to Scotland. Its objects were, first, to elucidate and amend those provisions in the National Health Service Acts which prohibited the sale of practices of doctors in the Service, and which compensated them for the loss of goodwill, in regard to partnerships of a mixed character. Secondly, the Bill implemented promises of amendment made to the medical and dental professions. Thirdly, it made certain minor administrative amendments, and, fourthly, it removed doubts regarding some of the provisions of the original Acts.

Great difficulty had been experienced in framing clauses dealing with partnerships and with the sale and purchase of medical practices, and even when these Bills became Statutes a good deal of discontent had remained concerning their effect. Mr. Bevan had appointed a committee under Mr. Justice Slade to examine those parts of the Acts which referred to partnerships, and he had completely accepted the report of this committee. The recommendations made in that report were provided for in the Bill. The Government wanted to encourage the formation of partnerships in the medical profession, believing this to be of advantage to doctors and to their patients. There would be as little interference with partnerships as possible. Lord Shepherd understood that there were only 50 "mixed partnerships" affected by the Bill, and the number of doctors concerned was only 150.

Part II of the Bill was simpler but of great interest. For some time the medical ranks had been agitated by a fear that under the Acts a full-time service might be established. Indeed, it was possible under the original Acts for Orders to be introduced into both Houses for the purpose of placing the medical service upon a full-time basis. Such a power had existed since the National Health Act of 1912. Mr. Bevan, however, had given a pledge in the House of Commons on April 7 that it would be made impossible by regulation to place the medical profession effectively on a full-time basis. Mr. Bevan had also said that the Government had no intention of establishing a full-time practice and that he would make this clear in an amending Bill.

Minor Amendments

Among other things the Bill referred to the tribunal which had power to take out of the Health Service those who must be disqualified. It was made up of a legal chairman, an executive council member, and a member of the profession concerned. The last member would be appointed by the Minister of Health, and at the suggestion of the medical profession would be taken from a panel representing the various interests of the profession concerned. The Minister had agreed to this.

Another point related to the costs of the establishment of local committees of doctors, chemists, and opticians. The professions concerned had suggested that the expenses of these committees should be borne out of block deductions from the fees to be paid to them. This had the advantage of maintaining the independence of the professions. The time in which doctors could make a claim for midwifery fees was extended to three months. Dentists would have the same rights as doctors in taking into hospitals as paying patients those of their clients who did not wish to be Service patients. The Minister held that as 95% of the population were in the scheme the number of persons among whom private practices could be built up was so small that they could receive the advantages of the hospital service without getting unduly in the way of those who were in the scheme.

In a great service relating to the hospitals questions of labour or remuneration were bound to arise. It was proposed, in

Clause 13, that the Conciliation Act of 1896 and the Industrial Courts Act of 1919 should be applied. A special tribunal of the Industrial Court would be appointed to deal with disputes arising out of remuneration or other conditions of service.

It was also intended to provide that men who were in the hospital service but desired to sever their connexion with it for a period should be enabled to do so but still remain in the superannuation scheme. It was desirable that medical officers should be able to take short-service contracts in the Colonial Medical Service while continuing to contribute to the Health Service superannuation scheme. Lord Shepherd went on to speak about clauses introduced at the request of outside organizations. District nursing associations and other bodies found much of their former work undertaken by hospitals and other authorities. The Bill would permit these bodies to hand over to the Health Services any property for which they no longer had any use but which existing trust deeds might have prevented them handing over. Clause 24, which applied to Scotland only, enabled children under 16 in that country to be treated on the initiative of parents or guardians as voluntary patients in mental hospitals. This would bring Scottish law into line with English law. Another provision, primarily a mental health matter, enabled a charge for board and lodging to be made to hospital patients who went out to work in the later stages of their recovery.

Lord Shepherd confessed that the Bill was somewhat disjointed. It was a list of small things promised, of little difficulties to be remedied, and of frictions to be eased. It was the first, but it would not be the last, National Health Service (Amendment) Bill. It was a matter for congratulation that the Government, having found that some adjustment was required, had taken early steps to bring it about. In the course of time as the Service grew the Government might have to call on Parliament to make other adjustments, but he believed that no attempt would be made to undermine the main principles on which the Acts were based. He moved that the Bill be read a second time.

One Shilling on Prescriptions

Lord LLEWELLIN said that had the promises which this Bill was to fulfil not been made the National Health Service might never have started. Although it was a minor Bill it did a major piece of work. Three years ago when the National Health Service Bill was discussed in the House of Lords statements were made that the partnership clauses would not work. Last time Lord Horder, Lord Moran, and Lord Webb-Johnson were all present in full cry. To-day those three noble Lords were conspicuous by their absence. From that it was clear that the medical profession agreed with the Bill.

He asked Lord Shepherd to tell the House whether the Government intended to use this Bill to effect the payment of one shilling per prescription which had been announced as part of the economy campaign, or whether that proposal would be contained in another measure. He had noted that old-age pensioners were to be exempt from the new charge, and he asked the Government to consider also the inclusion in this exemption of the disabled ex-Serviceman who needed medical supplies for his actual disablement.

Lord CLYDESMUIR said the parent Act had been applied to Scotland with little modification, and Scotland had the Health Scheme with all its mistakes, some of which were becoming evident. The medical services in Scotland were working under strain, and the House would assist those services by approving the Bill. The second part of the Bill provided that regulations should not require specialists to be employed whole-time. There was anxiety among specialists in Scotland about whether this clause was wholly watertight. A case had been instanced of a Scottish hospital in which as a result of administrative action, by advertisement, the door had been closed to anything but full-time employment of specialists.

Lord SALTOUN suggested that the provision for treatment in mental hospitals for children under 16, on the initiative of parents or guardians, might be an interference in Scotland with the boy's Common Law right. In Scotland a boy of 14 was considered capable of carrying on his affairs in all respects except ownership of a heritage.

The Earl of CORK and ORRERY, speaking of the proposed payment of a shilling on prescriptions, said that two men whom he had consulted had both asked why it was not arranged for a patient to pay the tax on his first visit to a doctor. That would limit the number of people going to see the doctor in the first place. It would cut down the mob in the waiting-rooms and would obviate the need for the doctor to go on prescribing coloured water. It would reduce the number of old women sitting there talking when all they needed was a Beecham's pill.

Lord KERSHAW replied briefly. He was unable to tell Lord Llewellin whether the shilling charge would be brought into the Bill. The matter was under consideration and was regarded as one which should be dealt with promptly. The suggestion with regard to disabled ex-Servicemen would be considered sympathetically by Mr. Bevan.

The Bill was then read a second time and was committed to a Committee of the whole House.

NURSES BILL

The Nurses Bill, which had its second reading in the House of Commons on October 21 (*Journal*, November 5, p. 1056), was considered on October 28 in committee.

Mr. LINSTEAD moved on Clause 1 the first of a series of amendments intended to give an assistant nurse the statutory title of "enrolled nurse" and to describe the relevant committee as the Enrolled Nurses Committee. He said that at the moment there was an anomaly because although these nurses were on a roll called the Roll of Assistant Nurses they had the right to call themselves nurses without using the word "assistant." Whether the House wished it or not many hospitals, particularly the hospitals for the chronic sick, would be staffed by assistant nurses. Anything which would help to bring these girls under the control of the General Nursing Council as assistant or enrolled nurses should be done.

Mr. BLENKINSOP sympathized with the object of the amendment. He said the Government did not regard the term "assistant nurse" as being a final choice. The Ministry had recently shortened the period of training for assistant nurses, and the time was a bad one to choose for suggesting a new name. It would be difficult for the average person to differentiate between enrolled nurses and registered nurses.

The amendment was then withdrawn.

Before the deputy chairman, Mr. BOWLES, called an amendment on Clause 2 in the name of Mr. WALTER ELLIOT to leave out "Standing Nurse Training Committee" and insert "Nurse Training Authority" he asked for an assurance that there was no more in it than a mere change of name. Otherwise he would have to rule the amendment out of order as conflicting with the money resolution already carried which authorized expenditure by standing nurse-training committees.

Mr. LINSTEAD moved the amendment and said that under the title first proposed the committees might be regarded as subordinate bodies of the regional board. That was not so, and the alternative name "authority" had commended itself to the Royal College of Nursing. Mr. BLENKINSOP was unable to accept this amendment, which was withdrawn.

An amendment by Mr. DIAMOND was also withdrawn, and Clause 3 was ordered to stand part of the Bill after Mr. Blenkinsop had said the Ministry was satisfied that under the Bill as it stood the General Nursing Council was entirely responsible for examinations and could standardize them throughout the country or authorize regional committees to carry them out on whatever conditions the Council considered necessary.

Training and Employment

Sir HUGH LUCAS-TOOTH moved to leave out subsection (2) of Clause 4. He said the purpose of the clause was to separate responsibility for the training of nurses from the employment of nurses. On second reading the House had not discussed how that purpose was to be carried out. He asked the Government to say how this scheme would work. Could a hospital do any training of nurses if it had not previously been approved by the nurse-training committee? If it could not, they might find that the scheme was what Mr. Bevan would describe as

muscle-bound. He also asked whether disbursements in respect of training would be made by the hospitals themselves or whether the nurse-training committee would be able to carry out a scheme within the walls of a hospital. He held it to be important that the actual training should be carried out completely by the hospital. Where there were two different bodies responsible for policy and payment there was danger of conflict. A hospital might require 50 nurses in training for the purposes of employment, but the nurse-training committee might decide that only 30 nurses should be in training in that hospital. How would that sort of question be resolved?

Mr. BLENKINSOP said the intention was first to issue a general indication of the category of expenditure which Mr. Bevan would regard as coming within the nurse-training fund. It would be possible to separate this from the general administrative expenses of the hospital, but some expenses which could be regarded as connected with nurse training could not reasonably be separated from general administrative expenses. To set out the categories of expenditure which the Minister regarded as being separately chargeable would be the first step. Categories of expenditure which Mr. Bevan would regard as coming within this section of the Bill would include the payment of sister tutors, expenditure on lecturers and others, and might also include certain types of building, such as provision for classrooms. Hospital management committees and boards of governors in framing budgets for nurse training would have regard to these specified types of expenditure, which would be open to variation from time to time. On the allocation of expenditure the Ministry hoped to get agreement in the regional committee. In the case of a new teaching centre which could also be used for other purposes of general hospital administration a regional committee might feel that the matter should not be included in the training budget. That was the type of question which would go to the Minister for decision.

Sir HUGH LUCAS-TOOTH asked whether the Government contemplated that the Minister should be able to divide up expenditure which was partly for training and partly for administrative expenses of the hospital.

Mr. BEVAN intervened to say it was perfectly clear that this was intended. The Bill was providing what was available already in the National Health Service to ensure that the academic side of medicine was kept separate and that the provision of apparatus on the secular side was the obligation of the Minister of Health.

The amendment was by leave withdrawn, and Clause 4 was added to the Bill.

Sir HUGH LUCAS-TOOTH moved on Clause 8 an amendment to provide that a finance committee should consist of not less than seven nor more than eleven members.

Mr. BLENKINSOP said the House would be undertaking responsibility which it had better leave to the General Nursing Council if it specified any precise numbers for the finance committee. He believed the general view was that it was desirable for the finance committee to be reasonably small. The amendment was withdrawn, and Clause 8 was ordered to stand part of the Bill. Minor amendments were made to Clauses 12, 13, 15, 16, and 19 on the motion of Mr. BLENKINSOP.

On Clause 21 Mr. BLENKINSOP secured the insertion of a provision that "chief male nurse" should mean a male nurse in charge of the male nurses employed in a mental hospital.

General Nursing Council

A new clause was added to the Bill to provide that membership of the Council and its subordinate committees should not involve disqualification from election to Parliament. Discussing the First Schedule, Mr. HOWARD moved to provide that one member of the General Nursing Council should be a member of the University Grants Committee. Mr. BLENKINSOP resisted the amendment and it was withdrawn.

Mr. BURDEN moved to provide that one member should be a nurse whose name was included in the supplementary part of the *Register* containing the names of nurses trained in the nursing of cases of infectious disease. He said the amendment had the support of the National Association of Local Government Officers. Mr. BLENKINSOP, in resisting the amendment, said he was glad to announce that owing to the decline in the need for fever nurses this part of the *Register* was becoming smaller. New admissions had fallen from 740 in 1946 to about 300 this year. The Government preferred to look forward to the early cessation of special training of this kind and would expect to find within the general training of nurses the necessary knowledge of infectious diseases. The amendment was then negatived.

Mr. SKINNARD moved an amendment to raise the representation of ward sisters on the General Nursing Council from one to two. Mr. HASTINGS supported the amendment and said that ward sisters required representation because they were engaged practically all their time, or ought to be, in nurse training. Mr. BLENKINSOP said it was wrong to assume that the representation of each particular viewpoint must be ensured specifically in the Bill. The amendment was negatived.

Mr. HOWARD thereupon moved that two members of the Council should be registered medical practitioners. He said the best results in nursing were obtained only where there was the closest working partnership between the medical man and the nurse. Mr. BLENKINSOP said in the past the Minister had always ensured representation of the medical profession. There was no intention to depart from that practice, but he preferred not to agree to the insertion in the Bill of the words proposed by Mr. Howard.

Dr. H. B. MORGAN said he failed to see why the medical profession should by statute be included on the General Nursing Council unless in the pending reform of the General Medical Council there was to be a reciprocal arrangement by which nurses would be included. He would tell the B.M.A. Council at its next meeting that he failed to see why the medical profession should ask to have representation on the General Nursing Council. The amendment was negatived.

Mr. HASTINGS then moved to provide that one member of the Council appointed by the Minister should be a person having experience in the nursing of cases of tuberculosis. He said nurses who had practical experience in nursing this disease would be of the greatest help to the General Nursing Council and indirectly to the Government in explaining what was wrong and in getting more nurses for this important class of case. Mr. BLENKINSOP said if it was not possible to meet the need in any other way he would be much inclined to accept the amendment. He hoped that increasingly nurses would gain experience in this field as part of their general training. He believed they would get experience of this field through the general representation of nurses, and he asked Mr. Hastings not to press the amendment. Mr. HASTINGS withdrew his amendment.

Further amendments to the First Schedule were moved and withdrawn and the Schedule as amended was agreed to. The Second Schedule (Standing Nursing Training Committees) was agreed to without amendment. Minor amendments were made on the motion of Mr. BLENKINSOP in the Third Schedule (Mental Nurses Committee). The Bill was then reported to the House with amendments.

Food Hygiene

Mr. SKEFFINGTON-LODGE on October 31 raised the question of food-poisoning and food hygiene. He said that between 1938 and 1947 there had been 3,270 outbreaks of food-poisoning in this country. In 1938 there were 156, as against 964 last year. There was reliable authority for the view that food-poisoning generally was at least three times what it had been before the war. Returns of the Registrar-General for the years 1937–46 recorded 246 deaths from this cause. Mr. Skeffington-Lodge calculated that one-half of the population had at least one meal a day away from home. Midday meals were served in schools to about two-thirds of the school population of the country. Since the war there had been two very hot summers, and many weaknesses on the food front had been exposed. The precautions taken in a cool summer would have to be looked at and revised, for experience showed that they were not adequate to deal with changed conditions and what might be a changing climate. He asked Dr. Summerskill whether she was satisfied that in the circumstances the powers of inspection which were available to her staff and to local authorities under the Food and Drugs Act were applied rigorously enough.

British standards of food hygiene were lower than in the United States and very bad indeed. He regretted that the model by-laws which had just been issued did not include among their excellent recommendations any reference to smoking. The preparation, handling, and serving of food should not be mixed with smoking. Customers should complain when utensils and personnel were unclean, when tea-towels were used for washing hands and cutlery and also for wiping table tops, or when washing-up water in kitchen premises was used over and over again, or when open displays of food were not properly protected from contamination.

Dr. EDITH SUMMERSKILL said that if the model by-laws recently distributed were adopted, and if the provisions made in them were carried out, it would be possible to eliminate most of these abuses. Unfortunately the public conscience was not sufficiently aroused to demand that there should be an alteration in the present practice. Outbreaks of food-poisoning were increasing; yet everyone knew that they were preventable. In fact the people had the cure in their own hands. She said that food which in the past had been thrown away was now stored, rehashed, and reheated, and so bacteria were given a new lease of life.

Mr. Skeffington-Lodge had quoted figures showing the number of cases since 1938, but he must remember that until that year food-poisoning had not been a notifiable disease. Only since then had public health laboratories supplying data on this subject been increased from 12 to more than 50. The country was more conscious of food-poisoning than in the days before 1938. The Central Council for Health Education, through the local authorities, had made an important contribution to the solution of the problem. Women's organizations also had done everything in their power. She appealed to housewives, if they saw the conditions which had been mentioned, to protest at the time and not to go to that shop again.

Salaries of Specialists

On November 1 Mr. CARMICHAEL asked the Secretary of State for Scotland if he knew of the decision of the Western Regional Hospital Board to increase the salaries of certain members of the medical staff, in some cases by more than 100%.

Mr. WOODBURN replied that the Western Regional Hospital Board had taken no such decision. They had recently made contracts with the doctors working in their hospitals providing for payment in accordance with the nationally agreed scales. These scales were settled by agreement between the Government and the medical profession last June and were based on the recommendations of the Spens Report, which was published and accepted by the Government in principle in June, 1948. All hospital authorities in the National Health Service had to observe these nationally agreed scales. No regional hospital board had any discretion in the matter.

The Services

Flight Lieutenant M. E. Fearnley, R.A.F., has been awarded the D.S.C. for great courage when he was flown to *H.M.S. Amethyst* on April 21, 1949, and joined her under heavy fire. With great skill and untiring devotion he rendered invaluable services to *H.M.S. Amethyst's* wounded.

Surgeon Lieutenant J. M. Alderton, R.N., has been posthumously mentioned in dispatches for outstanding courage and devotion to duty in *H.M.S. Amethyst* when she was fired upon by Chinese military forces while she was proceeding to Nanking on April 20, 1949, and subsequently when forced to remain for three months in the Yangtze River under the constant watch of shore batteries until she made her daring escape down the river on the night of July 30–31, 1949.

Surgeon Commander W. B. Taylor, R.N., and Surgeon Lieutenant W. L. Owen, R.N., have been mentioned in dispatches for outstanding courage and devotion to duty while serving in H.M. Ships *London* and *Black Swan*, respectively, during their attempts to assist *H.M.S. Amethyst* while under very heavy gun-fire on April 20–21, 1949.

Universities and Colleges

UNIVERSITY OF OXFORD

Corrected announcement

In a Congregation held on October 13 the following medical degrees were conferred:

B.M.—J. G. Widdicombe, E. A. D. W. Simpson, R. W. Torrance, *Nina A. Cohen.

*In absence.

UNIVERSITY OF CAMBRIDGE

In a Congregation on October 15 the degree of M.A. was conferred on Frank George Young, D.Sc., F.R.S., Sir William Dunn Professor of Biochemistry in the University. Professor Young has been appointed a member of the Council of the School of Veterinary Medicine until December 31, 1950, in place of Dr. Chibnall, who has resigned.

The Raymond Horton-Smith Prize for 1948–9 has been awarded to John Rupert Squire, M.D., for his essay on "The Relationship Between Horse Dandruff and Horse Serum Antigens in Asthma." *Proxime accessit:* Michael Braithwaite Paul, M.D., whose subject was "Thoracoscopy and Internal Pneumolysis in the Treatment of Pulmonary Tuberculosis."

UNIVERSITY OF LONDON

A special University Lecture on "Some Problems of Old Age" will be delivered by Dr. J. H. Sheldon at the London School of Hygiene and Tropical Medicine, Keppel Street, Gower Street, W.C., on Tuesday, December 1, at 5.30 p.m. The lecture is addressed to students of the University and to others interested in the subject. Admission is free, without ticket.

ROYAL COLLEGE OF PHYSICIANS OF LONDON

At a quarterly comitia of the College, held on October 27, with the President, Lord Moran, in the chair, Dr. E. C. Dodds, F.R.S., was elected Harveian Librarian, and Dr. C. F. T. East, Dr. N. B. Capon, Sir Horace Evans, Dr. J. G. Scadding, and Dr. T. F. Fox were elected Councillors.

The following were elected representatives of the College: Dr. C. M. Hinds Howell on the Committee of Management of the Conjoint Board; Dr. F. S. Langmead on the Central Midwives Board; the President, Sir Leonard Parsons, Dr. H. E. A. Boldero, Dr. W. G. Barnard, and Sir Allen Daley on the Standing Joint Committee of the three Royal Colleges; Dr. Hinds Howell, Dr. T. C. Hunt, Dr. J. C. Hawksley, and Dr. J. B. Harman on the Committee of Reference; and Dr. Hinds Howell and Dr. Harman on the Central Medical War Committee.

Dr. J. C. Broom was appointed Milroy Lecturer for 1951, his subject being "Leptospirosis."

The President announced that the Jenks Memorial Scholarship had been awarded to Barry Reid Wheeler, late of Epsom College.

MEMBERSHIP

The following candidates, having satisfied the Censors' Board, were elected Members of the College:

J. D. Aitchison, M.B., L. S. Antonoff-Lewis, M.B., P. E. Baldry, M.B., T. B. Binns, L.R.C.P., M. I. Bostock, B.M., D. P. Bowler, M.B., R. E. Britten Jones, M.D., P. H. Buxton, M.B., L. Capper, M.B., R. A. Chambers, B.M., D. Collins, M.B., Geraldine E. L. Cummins, M.B., R. R. De Mowbray, B.M., Captain, R.A.M.C., M. F. J. Donnelly, M.D., Margaret D. D. Dudley-Brown, M.B., J. Dundas, M.D., M. Y. El-Gammal, M.B., D. G. Evans, M.B., P. H. Friedlander, M.B., R. McK. Fulton, M.B., W. F. M. Fulton, M.B., J. M. Gardiner, M.D., I. Gordon, M.B., K. G. Green, M.D., R. Greenwood, M.B., J. H. Gubbin, M.B., D. B. Gunasekara, M.B., K. J. Gurling, M.B., E. J. Halliday, M.B., G. P. Hallwright, M.B., W. S. Hamilton, B.M., R. M. Hardisty, M.B., H. W. Holland, M.B., J. G. Holmes, M.B., Mary C. Holt, M.B., J. A. Irwin, L.R.C.P., A. D. M. Jackson, M.B., G. A. Jackson, L.R.C.P., S. L. O. Jackson, M.B., R. N. Johnston, M.B., H. E. M. Kay, M.B., R. J. Kernohan, M.D., J. D. S. Knight, M.B., D'A. Kok, M.B., M. H. Lessof, M.B., N. A. Lewtas, M.B., H. M. Lloyd, B.M., H. E. Magee, M.B., I. A. Magnus, M.B., R. H. Marten, B.M., F. B. Meade, M.B., J. B. Mitchell, M.D., J. K. Morgan, M.B., B. Mullally, M.B., N. A. Neville, B.M., L. L. Nussbaum, M.B., J. S. Oldham, B.M., D. N. Patel, M.B., R. A. Peebles Brown, M.B., E. R. Price, M.B., D. A. Pyke, M.B., J. Rankin, M.D., R. M. E. Seal, M.B., P. H. Shorthouse, L.R.C.P., Major, R.A.M.C., R. G. Simpson, M.D., A. L. Speirs, M.B., P. G. Swann, M.D., W. P. Sweetnam, M.D., M. G. Thorne,

M.B., N. A. Thorne, M.D., K. B. Thornton, M.D., W. F. Townsend-Coles, M.D., G. F. Vaughan, M.B., J. M. Walshe, M.B., P. Ll. Watts, M.B., J. Wedgwood, M.B., T. C. P. Williams, L.R.C.P.

LICENCES

Licences to practise were conferred upon the following 173 candidates (including 31 women) who had passed the Final Examination in Medicine, Surgery, and Midwifery of the Conjoint Board and who have complied with the necessary by-laws:

A. G. Ackerley, J. D. B. Andrews, Doreen R. Austin, G. R. Baji, J. P. E. Balbirnie, Barbara B. Ball, M. J. Ball, A. S. Basri, W. H. B. Begg, G. I. Benson, Peggy M. Birch, F. J. Birkett, B. Ll. Bisley, K. Böheimer, J. M. Bolton, R. K. Bowman, T. L. Pradbeer, Anna A. Braga, M. T. D. Braide, H. H. Broadhead, H. S. Brown, D. W. Bull, G. Canty, N. S. Capstick, Nancy I. Cardno, P. Chaudhuri, P. H. Cima, Brigid M. R. Clark, C. R. Cowan, J. L. Cowan, H. A. Cowie, C. C. Cox, J. S. Cox, C. C. Crampton, R. A. Crook. D. I. Davies, D. J. Davies, G. Davies, R. A. L. Davis, Ida J. Dawson, J. De Choisy, Marie T. M. D'Netto, R. P. G. Dickerson, A. K. Doust, B. W. Durrant, H. Duschinsky, Betty B. Dyer, Z. Elkes, Diana M. Elkington, B. D. Ellis, T. Lawford Evans, T. Leighton Evans, J. L. Facer, Dorothy Field, J. F. Filbee, R. H. P. Fitzpatrick, Alison M. M. Fleming, R. G. Forster, A. F. Franks, R. A. Garson, J. B. Gayes, H. R. E. Gillespie, J. E. Glover, J. D. Griffiths, J. J. Griffiths, G. E. Hanks, R. G. Hardwick, C. E. Hartley, S. K. Heywood, B. Hirst, C. McA. Hogg, T. C. Hopkins-Husson, P. T. Howse, R. T. Hughes, A. C. Hunt, P. J. Hynes, P. G. Jackson, Hilary M. K. Jenkins, W. P. Johnston, N. Jones, O. Ll. Jones, J. H. G. Jowett, Lucy E. Kaye, G. Kazantzis, Patricia M. Keefe, G. E. Kelly, S. M. A. Khadri, B. G. Kiddle, Barbara Kilpatrick, E. T. Knudsen, S. A. Kotz, R. G. F. Lampart, K. Lee, D. M. Lewis, P. E. Lewis, R. I. Lindfield, J. W. Lloyd, B. R. W. Lodge, D. H. Mackenzie, L. W. Mackey, I. E. J. McLauchlan, L. K. Manhajm, H. T. P. Martin, C. E. A. Mason, J. H. Mason, E. T. Mathews, D. C. Mayberry, B. Measday, Mary S. Medcalf, Maureen Meikle, J. A. Menon, F. G. Montfort, D. F. Morley, A. Morris, Elizabeth S. B. Nicoll, N. A. Nobbs, Lola E. Owers, Doreen B. Pallot, D. D. G. Parsons, R. E. H. Partington, Dina Patuck, J. H. Pauffley, P. M. Pawsey, A B. L. Peake, Dorothy J. Pearse, A. M. Platt, H R. Playfair, D. F. Potter, G. L. Powell, J. M. Price, D. C. E. Rabbin, Rebe S. Rainsbury, Olga M. Rampersaud, Stella J. E. Randall, F. G. Reader, M. Reckless, V. E. Rees, Joyce M. Richardson, J. J. Ridealgh, K. H. Ritchie, N. Rivers, J. S. Robertson, Yvonne K. Russell, M. Sackwood, J. G. Sahakian, C. M. Samuel, J. T. Scott, J. D. Shapland, E. W. Shepherd, E. A. D. W. Simpson, W. K. Slack, G. H. Smerdon, Margaret W. H. Smith, A. W. Sonia Gwendoline R. Stockley, W. H. J. Summerskill, A. S. Szurkawski, D. H. C. Thomas, Olwyn K. Thomas, O. L. Thomas, K. D. B. Thomson, N. A. Toes, A. Trueman, B. S. Turner, C. A. Unsworth, J. T. Wallace, D. E. Wallis, G. Walters, A. W. S. Webster, I. M. Wedgwood, B. L. Whitaker, R. Wilkins, S. W. Wilkins.

DIPLOMAS

Diplomas in Child Health, in Industrial Health, and in Physical Medicine were granted jointly with the Royal College of Surgeons of England to the successful candidates whose names were printed in the report of the meeting of the Royal College of Surgeons of England in the *Journal* of October 22 (p. 935).

Diplomas in Ophthalmic Medicine and Surgery were granted jointly with the Royal College of Surgeons of England to J. H. Owen-Flood and to the other successful candidates whose names were printed in the report of the meeting of the Royal College of Surgeons of England in the *Journal* of October 22 (p. 935).

Diplomas in Tropical Medicine and Hygiene were granted jointly with the Royal College of Surgeons of England to T. H. Work and to the other successful candidates whose names were printed in the report of the meeting of the Royal College of Surgeons of England in the *Journal* of October 22 (p. 935).

ROYAL COLLEGE OF PHYSICIANS OF EDINBURGH

At a quarterly meeting of the College held on November 1 with the President, Dr. W. D. D Small, in the chair, a Diploma of Fellowship was granted to Dr. J. J. R. Duthie (Edinburgh).

Diplomas of Membership were granted to Drs. J. H. Wright (Glasgow), W. H. Jopling (Rhodesia), T. S. Row (India), P. C. Mitchell (Aberdeenshire), R W. Crocket (Glasgow), R B. Baird (Uganda), W. H. Perry (Vancouver), J E. A Bartlett (Edinburgh), D. A. Gordon (London), R. F. Maggs (Capetown), H. N. Singh (India), P. C. Bhattacharjee (India), W. D. Munro (Edinburgh), B. Mukharji (India), G. I. Robertson (Stirling), W. J. Weston (New Zealand), M. W. M. Hadley (Edinburgh), M. J. Murray (New Zealand), K. S. Millingen (Sydney), J. N. Pohowalla (India), K. R. Keay (Anstruther), D. F. Gibbs (Edinburgh), A. M. Rudolph (Johannesburg), and Mary D. Smith (Glasgow).

INFECTIOUS DISEASES AND VITAL STATISTICS

We print below a summary of Infectious Diseases and Vital Statistics in the British Isles during the week ended October 22. [No. 42

Figures of Principal Notifiable Diseases for the week and those for the corresponding week last year, for: (a) England and Wales (London included). (b) London (administrative county). (c) Scotland. (d) Eire. (e) Northern Ireland.

Figures of Births and Deaths, and of Deaths recorded under each infectious disease, are for: (a) The 126 great towns in England and Wales (including London). (b) London (administrative county). (c) The 16 principal towns in Scotland. (d) The 13 principal towns in Eire. (e) The 10 principal towns in Northern Ireland.

A dash — denotes no cases; a blank space denotes disease not notifiable or no return available.

Disease	1949 (a)	(b)	(c)	(d)	(e)	1948 (Corresponding Week) (a)	(b)	(c)	(d)	(e)
Cerebrospinal fever	24	3	15	6	1	31	5	17	3	1
Deaths		—	—				2	—		
Diphtheria	92	9	30	3	7	153	16	29	12	7
Deaths	1	—	—	—	—	—	—	1	—	1
Dysentery	154	8	67	—	1	99	5	62	1	1
Deaths				—	—				—	—
Encephalitis lethargica, acute	2	—	—	—	—	1	—	—	—	—
Deaths		—					—			
Erysipelas			25	8	4			32	12	2
Deaths		—					—			
Infective enteritis or diarrhoea under 2 years				94					24	
Deaths	38	2	4	8	3	26	3	2	2	5
Measles*	1,052	40	37	119	53	5,303	86	100	37	116
Deaths†			—	—	—			—	—	—
Ophthalmia neonatorum	68	2	7	—	—	53	6	15	1	1
Deaths										
Paratyphoid fever	18	5	6 (B)	4 (B)	—	4	—	1 (B)	—	—
Deaths	—	—	—	—	—	—	—	—	—	—
Pneumonia, influenzal	317	13	5	1	—	426	24	2	1	1
Deaths (from influenza)‡	5	2	—	—	1	10	1	1	—	—
Pneumonia, primary			120	13				153	16	
Deaths	124	23		6	9	175	31		5	6
Polio-encephalitis, acute	29	2				4	—			
Deaths										
Poliomyelitis, acute	385	64	7	3	4	76	7	5	2	—
Deaths§	19	3				4	1			
Puerperal fever		—	5				—	12		—
Deaths										
Puerperal pyrexia‖	81	6	5	1	2	115	12	14	—	1
Deaths		—					—			
Relapsing fever	—	—			—	—	—			—
Deaths										
Scarlet fever	1,909	168	251	135	159	1,389	86	277	189	48
Deaths†			—	—	—			—	—	—
Smallpox	—	—	—	—	—	—	—	—	—	—
Deaths				—	—				—	—
Typhoid fever	6	3	—	3	1	13	1	4	3	—
Deaths	—	—	—	—	—	2	—	—	1	1
Typhus fever	—	—	—	—	—	—	—	—	—	—
Deaths				—	—				—	—
Whooping-cough*	834	26	53	44	46	2,163	120	95	52	17
Deaths	3	—	—	5	—	1	—	1	—	—
Deaths (0–1 year)	249	32	35	24	20	267	39	30	17	9
Deaths (excluding stillbirths)	4,085	651	507	161	100	4,371	710	534	158	104
Annual death rate (per 1,000 persons living)			10·2	10·0				10·8	9·9	
Live births	7,044	1121	844	411	214	7,270	1208	918	369	234
Annual rate per 1,000 persons living			16·9	25·5				18·5	23·1	
Stillbirths	172	18	32			193	24	25		
Rate per 1,000 total births (including stillborn)			37					27		

* Measles and whooping-cough are not notifiable in Scotland, and the returns are therefore an approximation only.

† Deaths from measles and scarlet fever for England and Wales, London (administrative county), will no longer be published.

‡ Includes primary form for England and Wales, London (administrative county), and Northern Ireland.

§ The number of deaths from poliomyelitis and polio-encephalitis for England and Wales, London (administrative county), are combined.

‖ Includes puerperal fever for England and Wales and Eire.

EPIDEMIOLOGICAL NOTES

Poliomyelitis

Notifications in the week ended October 29 were: poliomyelitis 347 (385), polio-encephalitis 20 (29). Figures for the previous week are shown in parentheses. This is the second week in which notifications have fallen.

In the following counties multiple cases were reported: London 55 (66), Bedford 2 (3), Berkshire 3 (6), Buckingham 4 (1), Chester 10 (8), Cumberland 2 (2), Derby 2 (5), Devon 3 (0), Dorset 3 (3), Durham 2 (3), Essex 26 (32), Gloucester 9 (12), Hereford 2 (0), Hertford 9 (6), Kent 16 (26), Lancaster 32 (25), Leicester 3 (13), Lincs, Lindsey 13 (7), Middlesex 17 (19), Norfolk 5 (8), Northumberland 2 (9), Nottingham 8 (9), Oxford 2 (1), Rutland 2 (1), Somerset 11 (10), Southampton 13 (13), Stafford 3 (12), East Suffolk 2 (1), Surrey 14 (16), East Sussex 9 (2), West Sussex 3 (2), Warwick 8 (9), East Yorks 2 (3), North Yorks 2 (3), West Yorks 50 (47), Glamorgan 7 (6).

Discussion of Table

In *England and Wales* there were decreases in the notifications of whooping-cough 41, acute pneumonia 37, acute poliomyelitis 33, polio-encephalitis 16, and dysentery 25, with increases in the incidence of scarlet fever 65 and measles 60.

The rise in the notifications of scarlet fever was contributed by the southern section of the country, while in the northern section there was a small fall in incidence. In the south-eastern and south-western counties a rise of 58 was recorded.

The largest rises in the incidence of measles were 44 in the Isle of Ely and 53 in Lincolnshire; the two chief centres of infection were Chatteris U.D. 57 and East Elloe R.D. 58. In the remainder of the country the trend of measles was unchanged.

The chief feature of the notifications of diphtheria was an increase of 7 in Durham. Only small variations occurred in the notifications of whooping-cough, notably decreases in Lancashire 32 and London 25.

Of the 18 cases of paratyphoid fever 12 were notified in Lancashire (Liverpool C.B. 6). A further 98 cases of dysentery were notified from the outbreak in Lancashire (Liverpool C.B. 28, Ince in Makerfield U.D. 21, Hindley U.D. 17, Abram U.D. 15); the only other important centre of infection was Glamorganshire 14 (Cardiff C.B. 10).

In *Scotland* there were decreases in the incidence of acute primary pneumonia 44 and whooping-cough 15. A fall of 13 was reported in the number of notifications of dysentery in Glasgow. Another 4 cases of paratyphoid B were notified in Lanark county.

In *Eire* there were increases in the notifications of scarlet fever 50, whooping-cough 13, and measles 11. The chief centres for scarlet fever were Dublin C.B. 70, Sligo, Sligo U.D. 12; for measles Waterford C.B. 32, Kilkenny, Kilkenny U.D. 30; and for whooping-cough Dublin C.B. 17 and Wexford, New Ross R.D. 16.

In *Northern Ireland* increases were recorded in the notifications of scarlet fever 15, whooping-cough 14, and measles 11. These trends were mainly influenced by the experience of Belfast C.B.

Statistical Review for 1947

According to Part II, Civil, of the Registrar-General's Statistical Review for 1947 the birth rate during the year at 20.5 per 1,000 total population was the highest rate since 1921. The marriage rate remained high at 18.6 per 1,000. Divorces were almost double the previous year's total, and 60,190 decrees nisi were made absolute during 1947 compared with a yearly average of 8,288 for the ten years 1936–45.

Week Ending October 29

Notifications of infectious diseases in England and Wales during the week included: scarlet fever 1,972, whooping-cough 749, diphtheria 101, measles 995, acute pneumonia 303, cerebrospinal fever 26, acute poliomyelitis 347, acute polio-encephalitis 20, dysentery 139, paratyphoid fever 14, typhoid fever 7, food-poisoning 130.

Medical News

Physicians to the King

Sir Horace Evans and Dr. D. T. Davies have been appointed Physicians to the King's Medical Household.

G.M.C. Election

Dr. O. C. Carter, of Bournemouth, has been elected a Direct Representative for England of the General Medical Council in place of the late Dr. John Bone. His term of office will be for five years, from November 8, 1949.

International European Society of Haematology

The Second International Congress of Haematology was held at Montreux on September 15–17. The presidents, Drs. P. Chevallier, of Paris, P. Lambin, of Brussels, and G. di Guglielmo, of Naples, welcomed haematologists from most European countries. Great Britain was represented by a small group, and Dr. W. Dameshek brought the greetings of the International Society of Hematology from the U.S.A. The secretarial work and the arrangements were in the hands of Dr. S. Moeschlin, of Zurich. The first day's discussion, on the haemolytic anaemias, was introduced by Professor Heilmeyer, of Freiburg, and Dr. Dameshek, of Boston, U.S.A. Antimitotic and cytostatic substances were considered, and a discussion was opened by Professor A. Haddow with a new hypothesis on the mode of action of nitrogen-mustard. Dr. L. M. Meyer, of Brooklyn, U.S.A., reported experience with folic acid and antagonists and mentioned especially his disappointment with aminopterin. Professor K. Rohr, of Zurich, finally pleaded for the continued maintenance of the classical terminology of blood cells, to which most people had become accustomed. Various demonstrations included phase-contrast photographs and marrow, liver, spleen, and lymph-gland biopsies.

Annual Meeting of University College Hospital

The annual meeting of University College Hospital was held in the Medical School on November 1. The chairman of the board of governors, Major-General Sir Harold Wernher, G.C.V.O., explained that it had been decided to hold the customary meeting in public since the introduction of the National Health Service. He said that the hospital now had supervision of St. Pancras Hospital, where 300 new beds with nurses' accommodation had recently been opened. The Tropical Diseases Hospital was also under their control. The beds in the private block had been reduced during the period under review in order to afford more accommodation for the nursing staff, and the out-patient department had been modernized. Sir Harold remarked that the National Dental Hospital, which was part of University College Hospital, had been overloaded with work since the advent of the Health Service, but the efficient teaching of undergraduates was being safeguarded. He added that the working of the Act had proceeded smoothly in the University College Hospital Group, but that costs had risen considerably, particularly in the last few weeks—for example, food bills had increased in price by 10%, and an extra £3,000 a year would have to be spent on streptomycin alone.

Memorial to Dr. Slome

Tributes were paid to the work of the late Dr. Irving Somerset Slome at a ceremony on October 26 at the London School of Chiropody when a memorial plaque was unveiled. Mr. R. Y. Paton, the present Director of Studies and President of the School's Governing Council, introduced Mr. R. W. Raven. Mr. Raven said that Dr. Slome had been one of the best teachers of anatomy in London. He had done a great deal in seeking the co-operation of hospitals in connexion with the training of students in chiropody. Mr. A. M. A. Moore, President of the Board of Registration of Medical Auxiliaries, then unveiled the plaque, and described how Dr. Slome, a South African by birth.

had been one of the most brilliant students at the London Hospital, where he qualified in 1935. His death at the early age of 36 was a great loss.

Homoeopathic Hospital Centenary

The London Homoeopathic Hospital marked its centenary by a reception on November 2 in the Great Hall of the British Medical Association, Tavistock Square. The King, who is patron of the hospital, sent a message, and the reception was attended by the Duke and Duchess of Gloucester, who were received by Mr. A. Clifton Brown, chairman of the management committee, and Sir John Weir, honorary consulting physician to the hospital. The Duchess was presented by a member of the nursing staff with a bouquet compounded of 50 varieties of flowers and plants from all of which homoeopathic drugs are regularly prepared. The hospital was established in 1849 by the English Homoeopathic Association in a small house in Golden Square. It comprised only 25 beds. In 1857 the freehold of the fine property at the corner of Great Ormond Street and Powis Place was acquired, and the new building was opened on May 12, 1857, the Lord Ebury of that day giving the inaugural address. The eastern part of the hospital is on the site of a house where Macaulay spent the years of his early manhood and wrote the first of his famous essays. It is intended further to mark the centenary by an appeal for funds to carry on homoeopathic research, education, and practice.

Cigarettes for the Disabled at Christmas

The Alexandra Musical Society is arranging to send boxes of cigarettes to disabled ex-Servicemen in hospitals, convalescent homes, sanatoria, and mental hospitals on Christmas Day. This scheme has been made possible through the generosity of the Rhodesia Tobacco Association. Medical superintendents or matrons who have not already applied for their hospitals to be included in the scheme should write at once to Mr. Ernest C. Thomas, Honorary Organizing Secretary, Alexandra Musical Society, Room 412, County Chambers, Martineau Street, Birmingham. The gifts are only for the disabled ex-Servicemen who will be patients in hospital on Christmas Day. The society also hopes to entertain 1,000 disabled ex-Servicemen at various Christmas parties. To enable these events to be carried out an appeal is made for £500. Donations will be greatly welcomed and appreciated. Her Majesty the Queen has honoured the society with a donation towards its work for disabled ex-Servicemen.

British Rehabilitation Methods in Greece

On a visit to Greece in March this year, which was arranged by the British Council in collaboration with the British Medical Association, Mr. St. John Buxton, senior orthopaedic surgeon at King's College Hospital, and Mr. A. E. Whiffing, technical inspector to the Ministry of Pensions limb-fitting centre at Roehampton, gave advice to Greek hospitals and artificial limb factories on British methods. The co-ordinating committee of the Greek Recovery Programme has now invited them to return, their expenses being paid by the Economic Co-operation Administration. Mr. St. John Buxton will leave for Athens on November 16 to give further advice on rehabilitation and to deliver lectures on orthopaedic surgery, and Mr. Whiffing has been seconded for three months to the co-ordinating committee to reorganize the artificial limb factories.

Wills

Dr. Brian Ingram Johnson, pathologist at Churchill Hospital, Oxford, who died in a car accident on March 19, left £50,043. Dr. James Barr, of Torquay, left £37,500; Dr. Robert Anderson, of Edgbaston, Birmingham, £13,310; and Dr. William Forbes McDonald, of Bristol, £1,194. Mr. Edgar Somerville, of Newton Abbot, Devon, left £40,976; Dr. James Galbraith, of Dingwall, £42,783. Professor John McGibbon, of Crieff, who was emeritus professor of midwifery and gynaecology, University of St. Andrews, left £5,249; Mr. Arthur Herbert Buck, of East Dereham, Norfolk, £4,391. Mr. Owen William Richards, of Bideford, Devon, formerly director of the Egyptian Government School of Medicine, left £72,109.

COMING EVENTS

Institute of Laryngology and Otology

Professor Geoffrey Jefferson, F.R.S., will deliver the annual address at the Institute of Laryngology and Otology, 330–332, Gray's Inn Road, London, W.C., on Friday, November 18, at 4.30 p.m. His subject is "The Acoustic Neuromas."

Liverpool School of Tropical Medicine

A reception will be held at the Liverpool School of Tropical Medicine, Pembroke Place, Liverpool, on Tuesday, November 22, at 7.45 for 8.15 p.m., on the occasion of the fiftieth anniversary of the opening of the school and presentations of the Mary Kingsley Medal.

Society of Medical Officers of Health

The annual dinner of the Society of Medical Officers of Health will be held at the Piccadilly Hotel, London, W., on Thursday, November 24, at 7 for 7.30 p.m.

Leeds School of Medicine

The fifty-ninth annual dinner of the past and present students of the Leeds School of Medicine will be held at the Great Northern Hotel, on Friday, November 25, at 7 for 7.30 p.m., when Professor J. W. McLeod, F.R.S., will preside. Professor H. S. Raper, F.R.S., Dean of the Faculty of Medicine of the University of Manchester, will be the guest of honour. Tickets may be obtained on application to the honorary secretary, at the Medical School, Leeds.

National Association for Mental Health

A one-day inter-clinic conference of the professional staff of child-guidance teams will be held at the Y.W.C.A. Central Club (Queen Mary Hall), Great Russell Street, London, W.C., on Saturday, December 3. The fee for admittance to the conference is 5s. (2s. 6d. for full members of the association), and applications should be sent to the conference secretary, Inter-clinic Conference, National Association for Mental Health, 39, Queen Anne Street, London, W.1.

Conference on Psychology

A conference on "The Outlook for Psychology" will be held at the Oxford House, Mape Street, Bethnal Green Road, London, E.2, on Sunday, December 4, starting at 10.30 a.m. Among the speakers will be Dr. W. Lindesay Neustatter, on "Psychology in the Clinic." Particulars may be obtained from the Oxford House.

Manchester Medical Ball

The Manchester Division of the B.M.A. and the Manchester Local Medical Committee have arranged a medical ball to be held at the Midland Hotel, Manchester, on Wednesday, December 7, when there will be a dinner at 7.30 p.m. and dancing until 1 a.m. Tickets (£1 5s. each) may be obtained from Dr. F. S. Catto, B.M.A. Regional Office, 33, Cross Street, Manchester, 2.

SOCIETIES AND LECTURES

Monday

LONDON UNIVERSITY.—At University College (Chemistry Theatre), Gower Street, London, W.C., November 14, 5.15 p.m., "*The Chemistry of the Transuranics and Other Newly Discovered Elements*," by Professor H. J. Emeléus, F.R.S.

MEDICAL SOCIETY OF LONDON, 11, Chandos Street, Cavendish Square, London, W.—November 14, 8.30 p.m., Discussion: "*Medical Biopsies*," to be introduced by Dr. R. Bodley Scott and Dr. R. B. Terry.

UNIVERSITY COLLEGE, Gower Street, London, W.C.—November 14, 4.45 p.m., "*Bacterial Toxins*," by Dr. W. E. Van Heyningen.

Tuesday

EUGENICS SOCIETY.—At Royal Society, Burlington House, Piccadilly, London, W., November 15, 5.30 p.m., "*Family Service Units for Problem Families*," by Mr. David Jones.

INSTITUTE OF OBSTETRICS AND GYNAECOLOGY.—At Postgraduate Medical School of London, Ducane Road, W., November 15, 3 p.m., "*Radiotherapy in Treatment of Uterine Malignant Disease*," by Mr. Malcolm Donaldson.

INSTITUTE OF DERMATOLOGY, 5, Lisle Street, Leicester Square, London, W.C.—November 15, 5 p.m., "*Psoriasis*," by Dr. F. R. Bettley.

ROYAL COLLEGE OF PHYSICIANS OF LONDON, Pall Mall East, London, S.W.—November 15, 5 p.m., "*Devices for the Protection of the Worker Against Injury and Disease*," Ernestine Henry Lecture by Dr. Donald Hunter.

ROYAL PHOTOGRAPHIC SOCIETY: MEDICAL GROUP.—At 16, Princes Gate, London, S.W., November 15, 7 p.m., "*The Importance of Quality in Medical Photography*," by Mr. E. V. Willmott, F.R.P.S.

Wednesday

HARVEIAN SOCIETY OF LONDON.—At 26, Portland Place, London, W., November 16, 8.15 p.m., "*Oesophageal Disorders in Children*," by Dr. W. P. H. Sheldon.

INSTITUTE OF DERMATOLOGY, 5, Lisle Street, Leicester Square, London, W.C.—November 16, 5 p.m., "*X-ray Technique*," by Dr. C. W. McKenny.

MIDDLESEX COUNTY MEDICAL SOCIETY.—At Clare Hall Hospital, South Mimms, Barnet, November 16, 3 p.m., meeting.

NORTH EAST LONDON CLINICAL SOCIETY.—At Prince of Wales's Hospital, Tottenham, N., November 16, 8 p.m., films and discussion on antenatal and obstetric subjects.

ROYAL INSTITUTE OF PHILOSOPHY.—At University College (Anatomy Theatre), Gower Street, London, W.C., November 16, 7.30 p.m., "*Hume and Kant and Their Attitude to Metaphysics*," by Mr. P. F. Strawson.

ROYAL INSTITUTE OF PUBLIC HEALTH AND HYGIENE, 28, Portland Place, London, W.—November 16, 3.30 p.m., "*Hygiene in the Factory*," by Dr. J. J. O'Dwyer.

ROYAL MICROSCOPICAL SOCIETY, B.M.A. House, Tavistock Square, London, W.C.—November 16, 5.30 p.m., ordinary meeting. "*Cytology of the Neurone in Experimental Poliomyelitis*," by Dr. G. M. Findlay and Mr. F. C. Grigg.

SOCIAL PSYCHOTHERAPY CENTRE, 7, Fellows Road, London, N.W.—November 16, 5 p.m., "*Active Psychotherapy*," by Dr. A. R. Redfern.

Thursday

BRITISH INSTITUTE OF RADIOLOGY.—At 32, Welbeck Street, London, W., November 17, 8.15 p.m., joint meeting with Section of Radiology, R.S.M., "*Chemical Factors Modifying Radiobiological and Radiotherapeutic Response*," to be opened by Dr. John Read.

DURHAM UNIVERSITY.—At Royal Victoria Infirmary, Newcastle-upon-Tyne, November 17, 5.15 p.m., "*The Study of the Living Cell*," Rutherford Morison Lecture by Dr. Arthur Hughes.

HONYMAN GILLESPIE LECTURE.—At Anatomy Theatre, University New Buildings, Teviot Place, Edinburgh, November 17, 5 p.m., "*The Surgical Management of the Malignant Colon*," by Mr. G. T. Mowat.

ROYAL MEDICAL SOCIETY.—At Royal College of Physicians of Edinburgh, Queen Street, Edinburgh, November 17, 7.15 for 7.30 p.m. President's annual dinner.

ROYAL SOCIETY OF TROPICAL MEDICINE AND HYGIENE.—At London School of Hygiene and Tropical Medicine, Keppel Street, London, W.C., November 17, 7.30 p.m., laboratory meeting.

ST. GEORGE'S HOSPITAL MEDICAL SCHOOL, Hyde Park Corner, London, S.W.—November 17, 4.30 p.m., lecture-demonstration: "*Neurology*," by Dr. D. J. Williams.

WESTMINSTER HOSPITAL SCHOOL OF MEDICINE, Meyerstein Lecture Theatre, Horseferry Road, London, S.W.—November 17, 5.30 p.m., clinico-pathological demonstration. Discussions: (1) "*Osteitis Fibrosa Cystica*," (2) "*Asthma*."

Friday

BRITISH INSTITUTE OF RADIOLOGY.—At 1, Wimpole Street, London, W., November 18, 8.15 p.m., joint meeting with Section of Radiology, R.S.M., "*Chemical Factors Modifying Radiobiological and Radiotherapeutic Response*," to be opened by Drs. Frank Ellis and L. A. Elson.

BRITISH TUBERCULOSIS ASSOCIATION.—At 26, Portland Place, London, W., November 18, 3.15 p.m., Presidential address: "*Tuberculosis Services in the Future*"; "*Tuberculosis Survey in Malaya*," by Dr. Andrew Morland; "*Tuberculosis Survey in Western Samoa*," by Dr. Brian Thompson.

FACULTY OF RADIOLOGISTS: THERAPY SECTION MEETING.—At Royal College of Surgeons, Lincoln's Inn Fields, London, W.C., November 18, 2.15 p.m., "*Modern Conceptions of a Dosage Unit for Radiotherapy*," by Dr. L. H. Gray.

INSTITUTE FOR THE SCIENTIFIC TREATMENT OF DELINQUENCY.—At St. George's Institute, Broadbent Street, Davies Street, London, W., November 18, 5 p.m., annual general meeting and address by the Earl of Feversham.

INSTITUTE OF LARYNGOLOGY AND OTOLOGY, 330–332, Gray's Inn Road, London, W.C.—November 18, 4.30 p.m., "*The Acoustic Neuromas*," by Professor Geoffrey Jefferson, F.R.S., annual address.

MAIDA VALE HOSPITAL FOR NERVOUS DISEASES MEDICAL SCHOOL, Maida Vale, London, W.—November 18, 5 p.m., clinical demonstration by Dr. D. McAlpine.

ROYAL INSTITUTE OF PHILOSOPHY, 14, Gordon Square, London, W.C.—November 18, 5.15 p.m., "*Everything*," by Mr. H. L. A. Hart.

ROYAL SANITARY INSTITUTE, 90, Buckingham Palace Road, London, S.W.—November 18, 2.30 p.m., "*The New Milk (Special Designations) Regulations*," discussion to be opened by Dr. W. A. Lethem (Ministry of Food).

UNIVERSITY COLLEGE, Gower Street, London, W.C.—November 18, 5.15 p.m., "*Transmission of Excitation in Nerve and Muscle*," by Dr. Bernhard Katz.

WEST LONDON MEDICO-CHIRURGICAL SOCIETY.—At South Kensington Hotel, 41, Queens Gate Terrace, London, S.W., November 18, 7.15 for 7.30 p.m., dinner meeting. "*The Story of Cortisone*," by Dr. W. S. C. Copeman.

Saturday

YORK MEDICAL SOCIETY.—At 17, Stonegate, York, November 19, 8.30 p.m., "*Ascertainment and Educational Treatment of Deafness*," by Professor A. W. G. Ewing and Mrs. Ewing.

APPOINTMENTS

CRAWSHAW, DONALD G., M.B., Ch.B., D.C.H., D.P.H., Medical Officer of Health, Peterborough.

DAVIES, J. O. F., M.D., D.P.H., D.R.C.O.G., Senior Administrative Medical Officer, Oxford Regional Hospital Board.

EAST ANGLIAN REGIONAL HOSPITAL BOARD.—*Assistant Chest Physician (Senior Registrar) in Cambridge Area*, J. B. Mitchell, M.D. *Assistant Chest Physician (Senior Registrar) in East Norfolk Area*, P. H. Sutton, M.D., M.R.C.P.

LYON, D. M., M.B., Ch.B., D.P.H., Deputy County Medical Officer of Health and Deputy School Medical Officer for Kent

SOUTH WESTERN REGIONAL HOSPITAL BOARD.—The following appointments have been announced: *Geriatrician, Bristol Clinical Area*, W. Hughes, M.D., M.R.C.P., D.T.M. *Assistant Plastic Surgeon, Frenchay Hospital, Bristol*, D. C. Bodenham, M.B., Ch.B., F.R.C.S.

BIRTHS, MARRIAGES, AND DEATHS

BIRTHS

Cooper.—On October 15, 1949, at Southmead Hospital, Bristol, to Flavia Cooper (nee James), M.B., Ch.B., D.A., wife of Ashley Cooper, a second daughter—Caroline.

Piney.—On October 29, 1949, to Gladys (née Fleet), wife of Dr. J. Piney, a son

MARRIAGE

Watson—Lowis.—On October 10, 1949, at Dunleer, Co. Louth, Eire, John Ernest Watson, M.B., B.Ch., to Margaret Sarah Lowis.

DEATHS

Bergin.—On October 25, 1949, at Clifton, Bristol, Frank Gower Bergin, M.R.C.S., L.R.C.P.

Brown.—On November 2, 1949, in London, John Claude Brown, M.D., of 16, Addison Gardens, London, W.

Burnett.—On October 7, 1949, Arthur Hill Burnett, M.R.C.S., L.R.C.P., of Bitterne Park, Southampton, aged 68.

Curry.—On October 28, 1949, at his home in Towning Place, Cross Hills, Keighley, Yorks, Alexander Curry, L.R.C.P.&S.I.

Davis.—On November 5, 1949, at a nursing-home, the Rev. George Brocklesby Davis, M.D., of 371, Cherry Hinton Road, Cambridge, formerly of C.M.S., India, and Ely, Cambridgeshire, aged 76.

Debono.—On October 12, 1949, Salvino Debono, M.D., of Malta.

Eason.—On November 2, 1949, at Nuffield House, Guy's Hospital, Sir Herbert Lightfoot Eason, C.B., C.M.G., M.D., M.S., F.R.C.S., aged 75.

Fairbairn.—On October 31, 1949, at 1, St. Mary's Grove, London, S.W., Donald Charles Fairbairn, M.C., M.B., B.S.

Ferraby.—On November 1, 1949, at 140, Broomfield Avenue, Worthing, Sussex, George Arthur Ferraby, M.R.C.S., L.R.C.P.

Lake.—On November 1, 1949, in a Bristol nursing-home, Richard Lake, F.R.C.S., aged 87.

Macdonald.—On October 29, 1949, at Edinburgh, Coll Macdonald, M.B., Ch.B., of Westcliff-on-Sea, Essex.

Macdonald.—On November 2, 1949, at 9, Fernielan Avenue, Edinburgh, Angus Macdonald, M.B., C.M., F.R.C.S.Ed., late of Southdean, Colinton Road, Edinburgh.

Muir.—On October 29, 1949, at Lealands, Box, near Stroud, Gloucestershire, Joseph Corbett Muir, M.D.

Nixon.—On November 4, 1949, at Forbes Fraser Hospital, Bath, John Hobart Nixon, M.D., of 25, Deansway, Chippenham, Wilts, aged 72.

Palmer.—On October 31, 1949, at Exmouth, Cadwallader Edwards Palmer, M.B., B.Ch., Colonel I.M.S., Ret., aged 68.

Palmer.—On October 19, 1949, at Torquay, Sydney Joseph Palmer, M.B.E., M.D.

Price.—On November 1, 1949, at a nursing-home, Leamington Spa, Horace John D'Arcy Gerard Price, M.R.C.S., L.R.C.P., formerly of Nuneaton, Warwickshire, aged 68.

Pridmore.—On November 3, 1949, at 5, The Terrace, Chale, Isle of Wight, John Walter Pridmore, M.R.C.S., L.R.C.P., late of Ryde, Isle of Wight, aged 79.

Rogers.—On October 28, 1949, in London, Norman Cumming Rogers, M.B., Ch.B., of 3, St. John's Wood Road, London, N.W.

Roper.—On October 17, 1949, at a nursing-home in Rochester, Kent, Charles Roper, M.D., D.P.H., aged 71.

Russell.—On October 26, 1949, at Southport Infirmary, John Cooper Russell, M.B., Ch.B., of 33, Balfour Road, Southport, Lancs.

Sevestre.—On October 13, 1949, at 38, Springfield Road, Leicester, Robert Sevestre, M.D., J.P., aged 81.

Shelley.—On October 12, 1949, at St. Bartholomew's Hospital, Rochester, Arnold Shelley, M.B., B.Ch., of Camden House, Chatham.

Shipton.—On October 31, 1949, at Briarfield, Milford-on-Sea, Hants, William Shipton, M.D., J.P., of Buxton, Derbyshire.

Watson.—On October 15, 1949, at 21, The Circus, Bath, Andrew Gordon Watson, M.D.

Young.—On October 25, 1949, at Torwood, Tomintoul, James John Taylor Young, M.D., aged 84.

Any Questions?

Correspondents should give their names and addresses (not for publication) and include all relevant details in their questions, which should be typed. We publish here a selection of those questions and answers which seem to be of general interest.

Treatment of Retinitis Pigmentosa

Q.—*Is there any effective treatment of retinitis pigmentosa? Has there been any further work published on the implantation of preserved placenta, which I believe was suggested by Filatov?*

A.—The inquirer must be referring to four articles which appeared in the *American Review of Soviet Medicine*, 1946, 3, No. 5: "The Treatment of Retinitis Pigmentosa with Intramuscular Injections of Cod-liver Oil" (p. 388); "Retinitis Pigmentosa" (p. 395); "The Implantation of Preserved Placenta in Retinitis Pigmentosa" (p. 397); "The Implantation of Preserved Liver in Retinitis Pigmentosa" (p. 398)—all by V. P. Filatov and V. A. Verbitska. There was also an article by Von Max Neuenschwander in *Ophthalmologica*, 1948, **116**, No. 4/5, 262, under the title, "Placentatherapie der Retinitis Pigmentosa," which might be consulted.

Placental implants beneath the conjunctiva for retinitis pigmentosa are being tried out in this country, but no one is yet in a position to give a firm opinion about the value of this treatment. The feeling among certain Continental surgeons is that temporary subjective improvement occurs in some cases but that the visual field is not increased. No statements are made about slowing or checking the progress of the disease. Filatov's facts are not carefully set out in his papers on this subject. Oestrone and cervical sympathectomy have also been tried, but without any appreciable effect. The writer's impression to date is that the claims made for all the therapeutic measures tried for retinitis pigmentosa are unconvincing.

Mortality from Bovine Tuberculosis

Q.—*I have seen it stated in the Press that bovine tuberculosis accounts for about 1,500 deaths per annum in England and Wales. Is there any statistical evidence to support this statement?*

A.—There is no statistical evidence, in the usually accepted meaning of the term, to support this statement. The number of deaths from bovine tuberculosis is an estimate based on a series of necropsies where bacteriological examination has been carried out in fatal cases of non-pulmonary tuberculosis to determine whether the infection was of human or of bovine origin. In 30% of the cases the origin was found to be bovine. In some of the fatal cases of respiratory tuberculosis the infection is, of course, also of bovine origin. From the evidence available the number of deaths due to the bovine type of disease has been calculated as one-third of the deaths from non-pulmonary tuberculosis. During the last three years for which the figures of deaths from non-pulmonary tuberculosis in England and Wales are available a large decrease has been reported. Deaths from non-pulmonary tuberculosis in the years 1946–8 numbered 3,482, 3,394, and 2,897, respectively. It is therefore likely that 1,500 deaths per annum is an overestimate of the present level of mortality from bovine tuberculosis.

Stilboestrol and Dysmenorrhoea

Q.—*Can prolonged administration of stilboestrol cause dysmenorrhoea? For the last four months a young woman has been treated for acne on her face by 0.5 mg. of stilboestrol diproprionate twice a day during the second week of the intermenstrual period. The condition of the skin has improved, but lately the patient has complained of dysmenorrhoea on the first day, otherwise her periods are normal. Are there any known ill effects of indefinitely continued treatment?*

A.—Stilboestrol, even in heavy and prolonged dosage, does not directly cause dysmenorrhoea. If given during the first half of the cycle in amounts large enough to inhibit ovulation it prevents dysmenorrhoea, because oestrogen-withdrawal haemorrhage is always painless. In the case quoted the dose is hardly enough to prevent ovulation—it might even be stimulating it in a woman who has previously had anovulatory cycles, and this would explain the onset of painful periods. On the other hand, the onset of dysmenorrhoea is more common at this age than at the menarche, and it may be unrelated to the treatment. Although the continuous administration of oestrogens has side-effects such as endometrial hyperplasia, irregular and heavy uterine haemorrhage, and pigmentation of the nipples, the intermittent treatment described is unlikely to do harm even if continued for several months.

Estimation of Erythrocyte Sedimentation Rate

Q.—*Could you give me an authoritative opinion on the correct procedure for estimating the erythrocyte sedimentation rate (Westergren method)? My own technique is as follows: To 0.5 ml. of 3.8% sodium citrate is added 2 ml. of fresh blood. The tube is set up immediately and read after one hour. Frequently two tubes are set up simultaneously, as I find that slightly different readings are obtained from each. The points I would like elucidated are: (1) Why do different readings occur? (2) Should the blood be allowed to reach room temperature before the tube is set up? (3) Should flocculation occur in the 3.8% solution of sodium citrate? (4) If slightly less than 2 ml. of blood is used, in what manner is the reading affected?*

A.—The questioner's procedure is identical with that of the writer, and is preferable to the method described in the answer in the *Journal* of March 17, 1945, because it is very difficult to fill a 2-ml. syringe without introducing air-bubbles, thus making an erroneous dilution. To take the questions seriatim: (1) In duplicate estimations variations of up to 10% should be expected, because it is impossible to set tubes exactly vertical and deviations of as little as one degree from the vertical should make an appreciable difference. (2) This is probably unimportant. Thermal equilibrium with the surroundings will be reached very quickly after the blood has been drawn up into the tube, which should not be in a draught or in a place where rapid changes of temperature occur. If duplicate estimations are made at different temperatures, considerable differences in readings occur. (3) No. If gross agglutination or flocculation occurs, then either the blood has started to clot or the patient is suffering from gross hyperglobulinaemia, the commonest cause of which in this country is myelomatosis. If, however, the question is intended to imply that flocculation occurs in the citrate without the addition of blood, then this should not normally occur; when it does it is nearly always due to the growth of fungal mycelium. This may be overcome by sterilizing the citrate in 25-ml. batches in universal containers and throwing away the contents as soon as they cease to be crystal clear. (4) The writer cannot answer this question, but believes that errors of 10% have surprisingly little effect.

Phonocardiography

Q.—*What exactly is phonocardiography, and has it any special advantages? Is it possible to make gramophone records of the heart sounds?*

A.—Phonocardiography is the graphic registration of heart sounds and murmurs. Its chief clinical advantage is that it provides a permanent record of the auscultatory findings. But it has other advantages: (1) it may resolve auscultatory doubts; (2) it serves to time heart sounds and murmurs against other electrical or mechanical events which may be recorded simultaneously; (3) it has proved helpful in analysing the nature of the heart sounds. Unfortunately, phonocardiograms are difficult to perfect technically, and many records being obtained at the present time are not entirely reliable. Again, the simultaneous recording of other events which aid the interpretation of the records presents other difficulties and necessitates more elaborate equipment. For this reason phonocardiography is unlikely to be used widely in this country for many years. Most cardiological units and research departments, however,

are studying it. Henriques made gramophone records of the heart sounds which could be played back to an audience. The results were poor, sounds and murmurs being grossly distorted and interference being considerable. His machine met with little enthusiasm in this country, and so far as is known is not being made now. It could certainly not be recommended for didactic purposes; auscultation could never be learnt from this device.

Post-herpetic Neuralgia

Q.—*Is there any method of preventing post-herpetic neuralgia? What domiciliary treatment can be used for this complication? Are injections of liver extract and vitamin B of value?*

A.—The prevention of post-herpetic neuralgia in the elderly appears to depend on treatment during the active stage of the zoster infection. The hypodermic injection of 0.5 ml. of pituitrin daily for 21 days from the onset of the infection has always proved successful in the experience of the writer. Large doses of vitamin B_1 by injection, sodium iodide by mouth, and more recently the antihistamines have also been used with some success.

"Allergy" and "Hypersensitivity"

Q.—*Can you distinguish between allergy and hypersensitiveness? To a question on lipstick dermatitis (July 16, p. 189), in which the inquirer talks about allergy, the answer carefully avoids the term and refers to hypersensitiveness. Is there any proof of hypersensitivity to the eosin colouring matter in lipsticks? Eosin belongs to the fluorescein series, and these substances are used, for example, on the conjunctiva for diagnostic purposes. Are there any corresponding cases of hypersensitivity to fluorescein?*

A.—Some years ago writers on this subject considered allergy and anaphylaxis as subdivisions of the main subject—hypersensitiveness. Allergy was often defined as hypersensitiveness occurring in man. At the present time the tendency is to consider anaphylaxis as a subdivision of the main subject—allergy—and hypersensitiveness becomes synonymous with "allergic state." Whichever terminology is used, "hypersensitiveness" is interchangeable with "allergy" or "allergic state" so far as human reactions are concerned. It is merely a question of using the term which the writer considers more generally acceptable in this country. Apart from this, "hypersensitiveness" is sometimes used in a wider sense than the word "allergy," which implies, at least, an antigen–antibody reaction in the tissue cells. Thus a person might be described as hypersensitive to misuse of the word "allergy," but not as allergic to it. The conservative mode of thought in this country is perhaps reflected in a continued preference for the word "hypersensitiveness," as distinct from the Americans' ready acceptance of the shorter and neater "allergy."

Proof of hypersensitivity to the eosin colouring matter in lipsticks has been obtained—see Sulzerberger, Goodman, Byrne, and Mallozi, *Arch. Derm. Syph., Chicago*, 1938, **37**, 597—and is now generally accepted. The writer has not come across any reference to fluorescein sensitivity.

Cancer of Bladder in Dye Workers

Q.—*What is the incidence of bladder carcinoma among aniline workers? Has aniline ever been held responsible for cancer in any other sites? An aniline worker, a patient of mine, has developed an osteosarcoma of the humerus with multiple bone secondaries and involvement of the chest.*

A.—Bladder tumours are known to occur among a high proportion of workers in the aniline dye industry, but they are not in general attributable to aniline as such. The substances generally held to be responsible are amino derivatives of naphthalene and benzidine. Such tumours have been observed and recorded in many different countries, including America, Russia, Japan, and the principal countries manufacturing aniline dyes in Europe. The actual incidence is not known, since many workers leave the factories before the tumour has developed and are not traceable. However, it is held by various observers that where working conditions are unsatisfactory almost all the workers in the department may be attacked, but at different times and in different forms. Very occasionally the kidney is also involved.

Primary tumours in other sites than the bladder and kidney cannot be attributed to work with dyestuff intermediates. The disease has been known for some 55 years, but no suggestion has ever been made that such tumours as bone sarcoma occur more frequently among workers in dye intermediates than among the general population. Experimental evidence exists that other amino derivatives of cyclic hydrocarbons can produce primary tumours in other sites—for example, liver, stomach, as well as bladder—but there is no evidence that such tumours arise in workers as an industrial disease.

Persistent Monilia Infection

Q.—*Two male patients have persistent monilia infection of the oral cavity. Treatment with aniline dyes, aluminium acetate, and vitamins has been most unsatisfactory, and reference to numerous books offers little encouragement. Can you advise?*

A.—Persistent monilia infection of the oral cavity usually suggests an unhealthy condition of the mucous membrane, since it is generally thought that moulds will not grow where the mucous membrane is healthy. In the majority of cases attention to cleanliness is the most important measure. In persistent cases where the condition does not disappear with attention to oral hygiene a search for possible systemic causes should be made. The disease is frequently associated with deficiency of vitamin-B complex, and in such cases large doses coupled with intramuscular liver injections may prove beneficial. The condition is also occasionally seen in diabetics, and it is therefore advisable to exclude any diabetic tendency. For local application gentian violet is, of course, the standard treatment, but silver picrate preparations are equally effective and do not have the undesirable staining properties of the aniline dyes. Boric acid solutions are of little value.

NOTES AND COMMENTS

Acute Nephritis and Sweating.—Dr. CHARLES ROMER (Worcester) writes: From time to time the answers in "Any Questions?" must have surprised your readers and have made them wish that the replies were not anonymous. In the *Journal* of October 1 (p. 767) the hardy annual of whether to nurse a case of acute nephritis in blankets is revived. As the General Nursing Council still expects this (together with an intimate knowledge of leeches, wet and dry cupping, etc.) is it not time, Sir, to ask that the subject should be thrashed out and a really authoritative statement issued? Your answer states: "The General Nursing Council is acting in this matter with the full approval of medical authority. The scientific principle involved is allied to the known fact that patients suffering from this disease tend to sweat excessively." The answer continues very logically that such patients are more comfortable in blankets, and for rheumatic fever everyone is in agreement. No mention is made of the diaphoretic action, which must be taken as approval of such measures being, as Dunlop and Davidson in their *Textbook of Medical Treatment* (1949, Edinburgh) put it, "now generally abandoned." The crux of the matter is, then, do patients with acute nephritis "tend to sweat excessively"? No mention of such sweating is made in Price, Savill, Sheldon, Paterson, or Ellis's Croonian Lectures (*Lancet*, 1942, **1**, 34 and 72). None of the medical sisters asked, nor the writer, can recall having seen this alleged sweating. It is of interest to think that there can be such a wide divergence of opinion over a point that can be settled by simple clinical observation. The writer can see no reason for continuing to use blankets, but would beg for really authoritative views, lest another baby be thrown away with the bath water.

All communications with regard to editorial business shou'd be addressed to THE EDITOR, BRITISH MEDICAL JOURNAL, B.M.A. HOUSE, TAVISTOCK SQUARE, LONDON, W.C.1. TELEPHONE: EUSTON 2111. TELEGRAMS: *Aitiology, Westcent, London.* ORIGINAL ARTICLES AND LETTERS forwarded for publication are understood to be offered to the *British Medical Journal* alone unless the contrary be stated.

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